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UP JOURNAL OF OPHTHALMOLOGY

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Cover Photo

Stereoscopic Fundus Photograph of Glaucomatous Optic Neuropathy.

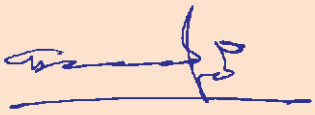
Courtesy : Prof. Tanuj Dada, AIIMS, New Delhi

Dear Dr Shalini Mohan,

It gives me immerse pleasure to see the UP state ophthalmological society growing by leaps & bounds. The website is fully functioning and is updated by efforts of Dr. Mohita Sharma. The midterm of UPSOS is being hosted by Dr. Amit Patel at Ambedkar Nagar and excellent scientific program has been made by chairman scientific committee Dr. Deepak Mehta.

This has been possible with team work positive attitude and untiring efforts of the executive committee. I feel elacted to introduce the second issue of the UP Journal of Ophthalmology and wish the editor Dr. Shalini Mohan and her team a great success in coming future.

Wishing you all a great reading experience.



Dr. Kamaljeet Singh, MS

President, UPSOS
Professor & Head, Department of Ophthalmology
MLN Medical College, Allahabad

Job Opportunity or Corporate Encroachment ?

Dear Friends,

"The opportunity is everywhere. The Key is to develop a vision to see it!!!!!!"

The present ophthalmologist is often at crossroad of his career and is confused to choose his career option even after acquiring reasonable surgical skills and qualifications, hence starts a race to excel commercially and make a name in fraternity.

Setting up an eye hospital is a herculean task and running it up with the pace of present day trickles sweat from the spine further.

Here, comes the introduction of corporate. Corporate hospitals give best working atmosphere, best of tools, invest in marketing and offer lucrative salary.

But do we know our commercial worth?

What's the grey line between over worked & exploitation ?

What's the Job security ?

This needs to be understood very well by each of us and career option should be weighed in all terms, may it be brand and amount of work/surgical exposure; also its commercial worth.

There is space for all formats of ophthalmic practice and we must access our academic & surgical strengths along with the commercial worth to choose our career option wisely.

"If you have got an amazing opportunity and you are not sure about grabbing it, figure out to do it later."

I hereby take the pleasure to introduce our second issue of UP Journal of Ophthalmology. I thank the President Prof Kamaljeet Singh, Secretary Dr Mohita Sharma, Joint editor Dr Ram Yash Yadav, Associate editors, editorial board and executive committee for whole hearted support.

Happy reading for all of us.

Warm regards

Dr Shalini Mohan
MBBS (Gold Medalist), MS, DNB, MNAMS, FCGP
Editor, UP Journal of Ophthalmology
Associate Professor
Chief Glaucoma, Cornea Services & Eyebank
Department of Ophthalmology, GSVM Medical College, Kanpur (UP)
Vice President: KOS
Ex Senior Resident Dr.R.P. Centre, AIIMS, New Delhi
Ex Consultant, Sir Ganga Ram Hospital, New Delhi.

Dear Members,

Greetings!!

We started the new term late last year with a lot of enthusiasm with the slogan "UPSOS for academics". This journal is playing a very vital role in this regard. I congratulate Dr Shalini Mohan and Dr Ram Yash Yadav for this.

Ophthalmology is advancing very rapidly with new technology knocking at our doors every day. Phacoemulsification is now a "refractive cataract surgery" With the advent of premium intraocular lenses. Our patients definitely deserve the best visual outcomes after cataract surgery.The journal is a step towards helping our members give to their patients the best, which they deserve. With advancing technology and techniques it is sometimes difficult to decide as to what should be the preferred practice patterns despite there being enough data on the internet to read. Panel discussion which is an important feature in this journal is the best way to help the readers decide what practices to follow as it brings our the differing views of multiple ophthalmologists on the same subject. This journal has very well incorporated this. And this needs to be the pattern of scientific programs in all our conferences too in the form of discussions and debates. This is the change that UPSOS intends to bring.

So happy reading and happy contributing!!

Mohita Sharma

Dr Mohita Sharma,
General Secretary, UPSOS

Save the Date for Next UPSOS

Moving Beyond Visual Acuity- Visual Quality and Stereoacuity

Jeewan S. Titiyal, MD **; Manpreet Kaur,** MD
Cornea, Cataract & Refractive Surgery Services,
Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India

Modern-day phacoemulsification is increasingly becoming akin to refractive surgical procedures, wherein a perfect visual outcome is expected in the postoperative period by the patient as well as the surgeon. To keep pace with increasing patient expectations, a wide range of premium multifocal IOLs are continually being developed with the aim of providing true spectacle independence at all distances. Conventionally, the success of the new IOLs are assessed in terms of visual acuity, with a 20/20 vision established as a benchmark parameter to evaluate the outcome of any lens.

However, the mounting evidence of 20/20 yet unhappy patients points towards the fallacy of using visual acuity as a sole outcome measure to assess IOL function. With more and more patients opting for multifocal IOLs, we also come across ever increasing reports of dissatisfied patients experiencing dysphotic symptoms and compromised visual quality.[1]

It is the need of the hour to shift our focus from visual acuity to more comprehensive parameters of visual function, encompassing visual acuity, quality and stereoacuity. We live in a three-dimensional (3-D) world, and with technological advancements 3-D is increasingly being integrated in the day to day life in the form of cinema, graphics, gaming as well as printers.[2] Conventional IOLs have focused on the correction of lower order aberrations only. However, in recent years, researchers are increasingly focusing on improving visual quality with better aspheric designs aimed towards reducing the higher order aberrations. Various aberrometers help to quantitatively assess the visual quality and higher visual function parameters, and these investigations help in a more comprehensive evaluation of visual function after implanting an IOL.

We have evaluated visual quality and stereoacuity after bilateral implantation of extended range of vision intraocular lens in patients undergoing phacoemulsification.[3] The extended range of vision (ERV) IOLs (TecnisSymfony, AMO) are the newer generation multifocal IOLs that aim to provide good visual acuity along all range of distances.[4] These IOLs do not result in the creation of two or three discrete foci as in conventional bifocal or trifocal IOLS; instead, they provide an elongated depth of focus and are associated with minimal dysphotic symptoms and optimal patient satisfaction. The IOL has an aspheric anterior surface to compensate for corneal spherical aberrations and an achromatic diffractive surface to correct chromatic aberrations. This results in an increase in retinal image quality without affecting the depth of focus.[5]

We observed a near stereoacuity of 30 seconds of arc or better in all our patients; of these, 80% cases had a perfect near stereopsis of 20 seconds of arc. Distance stereopsis of 100 seconds of arc or less was achieved by 82% cases, though fine distance stereopsis of 60 seconds of arc was achieved by only 36% cases.[3] The stereopsis correlated well with patient satisfaction and visual quality as assessed by ray tracing aberrometry, emphasizing the significance of a normal binocular interaction in assessing the quality of vision to ensure optimal patient satisfaction. There is a paucity of literature describing the normative values of stereopsis in older age group, though a worsening of stereoscopic threshold with age and in pseudophakia has been reported in few studies.[6] Our results of near stereoacuity after ERV IOL implantation were better as compared to previous studies with bilateral multifocal or monofocal IOL implantation.[7,8] This was the first study evaluating distance stereoacuity after bilateral multifocal implantation and further highlighted the need to integrate these parameters in routine postoperative evaluation.

Visual quality is conventionally assessed using aberrometry in terms of modulation transfer function (MTF), Strehl ratio and higher order aberrations (HOA). Modulation transfer function (MTF) is a measure of contrast and the Strehl ratio is a measure of the intensity of the image brightness, with an MTF and Strehl ratio of 1 signifying a perfect optical system. In recent years, aberrometry is being adopted as an additional investigative tool especially in postoperative assessment after multifocal IOL implantation. However, there is a lack of agreement regarding the specific cut-off values for these parameters and there exists wide variability between different aberrometer systems as well.[9] We observed good visual quality

as assessed by the ray tracing aberrometer and our results were in agreement with previous studies.[10,11] We considered 0.5 µm as the upper limit for coma, trefoil and spherical aberrations and all our cases had HOAs less than 0.5 µm.

There has been a quantum leap in IOL technology over the years and we have progressed from monofocal IOLs to bifocals, trifocals and now, ERV IOLs. In our clinical experience with ERV IOLs, we observed excellent visual acuity along all range of distances. Excellent near and distance binocular interaction was observed with ERV IOLs. Moreover, the stereoacuity correlated well with patient satisfaction and visual quality, highlighting the significance of normal binocular interaction in achieving optimal outcomes. Our study highlights the importance of routinely assessing binocular interaction with all IOLs, especially multifocal IOLs. Binocularity and depth perception are essential components of visual quality and may even act as markers for patient satisfaction. With improving technology, our focus should be to not only provide an optimal visual acuity across all range of distances but also an optimal visual quality with excellent binocularity.

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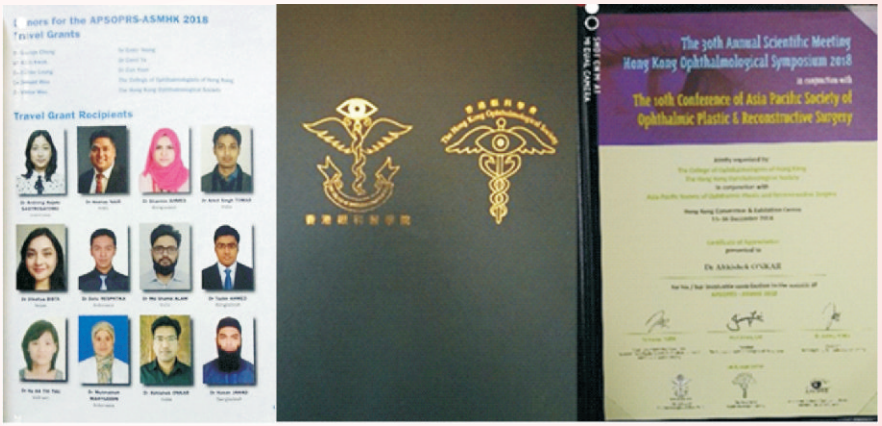
Congratulations

Dr Abhishek Onkar, ***(UPSOS—O-04)*** *Assistant Professor & Officiating Head, Department of Ophthalmology, ANIIMS, Port Blair*

For APSOPRS-ASMHK Travel Award, awarded by Asia-Pacific Society of Ophthalmic Plastic & Reconstructive Surgery (APSOPRS) on 16.12.2018 for scientific poster presentation in the conference.



Panel Discussion on Managing Cataract Surgery

UPSOS Correspondent : **Mohit Khattri**
Consultant, Regency Hospital Ltd, Kanpur

Expert Panel :

Amit Porwal (AP)
DO, FAEH, FLDR
Director, Sanghvi Nethralaya, Belagavi
Member AIOS-ARC, Central Zone

Chetan Anand (CA)
MS, FCED
Netradhama Super Speciality Eye Hospital, Mysore

Jatinder Wahi (JW)
MS, FCLI
Medical Director
Greens Eye Hospital, Lucknow

Vipin Sahni (VS)
MS, MBA
Medical Director
Kaushalya Devi Eye Institute. Pilibhit

People in different parts of country follow different protocols to manage the cataract surgeries done at their centres. We selected a panel of experts to know their unbiased responses on how they manage the surgeries at their centres in different formats of practice. Here are the excerpts of the responses. We hope that the readers would benefit out of this compilation and frame their own way to manage cases to give the best visual outcome to their patients.

Q.1 : What set of pathological investigations you advise your patient prior to routine cataract surgery?

AP: I get Physician fitness for all my cataract surgery patients, we have a in house full time physician. Random Blood Sugar in non-diabetics and FBS / PPBS in diabetics. Cut off for Random and PPBS is 160mg% and that for FBS is 120Mg%. Also we get HIV and HbsAg done.

CA: I get the following tests done:

- Complete blood count+ESR
- Random/ fasting & post prandial blood sugars (based on patient has h/o diabetes or not
- ECG
- HbsAg
- HIV
- In cases with any ocular inflammation history/h/o Rheumatoid arthritis etc, C-Reactive protein titre (not done as a routine in all patients)

JW: Keeping in mind of today's NABH guidelines and medicolegal scenario I ask for following investigations.

a- Blood Sugar Fasting & PP.

b- Conjunctival swab

c- Physical checkup by MD Medicine

d- HIV & HbsAg status.

VS: I order for Fasting and PP Blood sugar, HIV, HBsAg and cardiac clearance in selected cases

Q.2: Do you give topical antibiotics prior to cataract surgery? If yes, then which drug and what regime?

AP: Yes I do give topical antibiotics in combination with NSAID two days prior to surgery. I give a combination of Moxifloxacin and Ketorolac eye drop 4 times a day two days prior to surgery.

CA: No

JW: I routinely use moxifloxacin eye drops 1 drop 04 times a day, starting 02-03 days before surgery.

On the day of surgery - 04 drops regime- 1st drop as the patient is received in the hospital, 2nd drop when the patient changes his dress, 3rd drop when the patient lies on OT table , 4th drop before start of surgery

VS: I use intracameral antibiotics (moxifloxacin) but not prior to it

Q.3: How do you use Povidone Iodine (Betadine) in your cases?

AP: I use Povidone Iodine eye drops (Aurodone 5% e/d by Aurolab) 5 minutes before the surgery, I instil two drops in the eye. Also use 5% povidone Iodine skin antiseptic solution to clean the lids and the external area around the eye before surgery.

CA: As eye drops-

- 1 drop of 5 % povidone Iodine eye drops at least 20 minutes before surgery (after instilling proparacaine eye drops)
- 1 drop again before painting the eye- then flushed with 10 ml BSS

Regular painting of eye to be operated with Povidone Iodine solution (twice)

JW: Paint the eye before draping with betadine Instillation of 02% betadine in the culdesac for standing 03 minutes.

VS: Betadine skin paint in pre op room, second paint on OT table and a drop of betadine in conjunctival sac after speculum is on which is thoroughly flushed after 60 seconds prior to making incisions. Also a mandatory step is to scrub the lid margin in bud soaked with betadine prior to putting drape.

Q.4: Do you use Lignocaine jelly for your topical phaco cases or drops are sufficient for you?

AP: I use one drop of Paracaine eye drops two times at 2 minutes interval about 10 minutes before surgery. I also supplement later with 0.1 ml Intracameral Lignocaine (Oculan).

CA: Only topical 4 % lignocaine as eye drops before surgery. I do not use jelly.

JW: I do not use Lignocaine jelly but I use Topical drops, 01 drop 03 times before starting the surgery.

VS: Only proparacaine drops suffice.

Q.5: What are the indications for peri/retrobulbar block for you in phaco surgery?

AP: Local anaesthesia is preferred when the patient is very uncooperative, anxious, obese short necked, claustrophobic, asthmatic and when I am dealing with a complicated cataract surgery like Subluxated cataract, mature intumescent cataract with very shallow anterior chamber.

CA: Topical is preferred, but yes, sometimes local anaesthesis is required.

- Most common indication for me is hard of hearing patient as I believe that there should be a continuous verbal communication between surgeon and patient during surgery, especially in steps where patient may experience pain/pressure/burning (E.g. during instillation of intracameral lignocaine, during IOL insertion). If such steps are done without the patient anticipating discomfort, there may be a startle reflex with head movement/ strong bells
- When patient requires sedation during surgery (such as in very apprehensive patient, uncooperative patient etc.) as the bells phenomenon associated with sedation can be troublesome during surgery
- Nystagmus
- Very intumescent cataracts

I usually prefer Subtenon's anaesthesia with blunt cannula in such cases to peri/retrobulbar block with sharp needle

JW: a- When patient is really apprehensive.

b- When I am apprehensive for the patient (VIP Syndrome).

c- Sunken eyes

d- Non- Co-operative personality

VS: I prefer block with combined surgeries, subluxated cataracts, non dilating pupils, Nystagmus, extremely un co operative or apprehensive patients.

Q.6: Do you change the phaco tip after each case? If no, then do you sterilise it in between the cases?

AP: Yes I do change not only the tip but also the Phaco probe after each case. I use fresh sterile autoclaved Phaco probe for each case.

CA: Yes. Tip and sleeve changed after every case, machine is primed & tuned.

JW: I have a set of 05 Phaco tips and Sleeves which I change in all cases and then go for flash sterilization if need may be for the further cases.

VS: Tips and sleeves are definitely changed and flash

autoclave is used in long sessions.

Q.7: Do you change the blades after each case?

AP: A big YES. I use new disposable blades for each case, I never reuse them.

CA: Yes. Single use disposable blades in every case.

JW: Yes, I do change my MVR and 2.2 keratome in all the cases.

VS: Fresh blades in each case. A strong recommendation.

Q.8: What's the routine visit schedule of your post ops after a routine phacoemulsification?

AP: My routine visit schedule after cataract surgery is – 4 hours after surgery, then at 1 week and then at week 4 from the day of surgery. After that I tell the patient to get their eyes checked after 4 months for a routine check up.

CA: First day post OP and the 15th day post op.

JW: I insist for following schedule as much as it is possible, for post-op.

First visit - next day after surgery

Second Visit- Third day after the surgery.

Third Visit- Seventh day after the day of surgery.

Fourth Visit- One month after surgery

VS: I see the patients immediately after the theatre, next day, then weekly till 4 weeks post operatively.

Q.9: Till how long you give topical antibiotics post operatively to your patients routinely?

AP: 2 wks

CA: 15 days.

JW: Moxi - Prednislone combination eye drop 01 drop 04 times a day for 15 days, followed by tapering dose of 03 drops, 02 drops a day for consecutive next 02 weeks.

VS: 3 weeks

Q.10: Till how long you give topical steroids post operatively to your patients routinely?

AP: I give weekly tapering dose of steroids over 6 weeks.

CA: 6 weeks, weekly tapering regimen is followed

JW: Moxi - Prednislone combination eye drop 01 drop 04 times a day for 15 days, followed by tapering dose of 03 drops, 02 drops a day for consecutive next 02 weeks.

VS: Prednisolone drops for 3 weeks and then Loteprednol for next 3 weeks

Q.11: Do you use topical NSAIDS in pre/post op regime routinely? If Yes, then How?

AP: Postoperatively I use Nepafenac eye drop 3 times a day for 4 wks. Pre operatively I already mentioned that I use ketorolac eye drop 4 times a day for 2 days prior to surgery.

CA: Pre operatively, I do not use any topical NSAID routinely. Post op I give topical NSAIDs for 30 days. Presently using Nepafenac eye drops 3 times per day for 30 days

JW: Yes, I use flurbiprofen eye drops , 01 drop 04 times a day-03 days before surgery and I continue it for 01 drop 04 times a day for 15 days after surgery.

VS: I use NSAIDS only in eventful surgeries, pediatric cataracts and Diabetics

Q.12: After how many days post op do you give pseudophakic correction?

AP: At 4 weeks post op visit I give pseudophakic correction.

CA: 15 days post op

JW: I give the final refreractive correction after 01 month of normal phaco surgery.

VS: Minimum of 4 weeks I think it takes to have a stable refraction after cataract surgery

At the end, we personally feel that after reading, the practitioner must be able to frame some practical guidelines in his or her practice regarding the use of multifocal IOLs.

Wishing you all the best!

Mohit Khattri

Congratulations

Dr. (Prof.) Manav Deep Singh, *(UPSOS—S-B4)* Glaucoma Services, Dr. RML Hospital, New Delhi

For being elected as Library officer of Delhi Ophthalmic Society, 2019-2021 and as Treasurer of Glaucoma Society of India 2018-2020. Also Congratulations for the Best poster runner up award in GSI conference 2018.

Sutureless Extraocular SFIOL Technique

Shobhit Mehrotra, MS; **Sonal Sah Mehrotra,** MS; **Shri Nath Mehrotra,** MBBS, DOMS
Dr. Shivnath Memorial Eye Centre, Etawah

Purpose : To describe a novel technique of SFIOL fixation

Introduction: Conventional SFIOL fixation comes with a large number of challenges ranging from exteriorization of haptics to their fixation. There are many methods to exteriorise the IOL haptic. [1,2,3] These problems can be tackled with a modification in the current technique, thus helping the surgeon fix the SFIOL without sutures, flaps and glues. This technique was first described by Dr. Prabu Baskaran, Arvind Eye Institute, Pondicherry.[4]

Method: 3 & 9 o clock position are marked. Following conjunctival peritomy, a 23G MVR blade is used to make two partial thickness, 3mm linear scleral tunnels at the marked meridian 1.5 mm away from limbus.

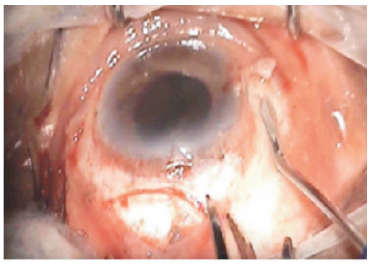


Figure 1:

The tunnels are created in an anticlockwise direction. Anterior vitrectomy is performed and an AC maintainer placed. A 5.5mm self sealing sclerocorneal wound is made.

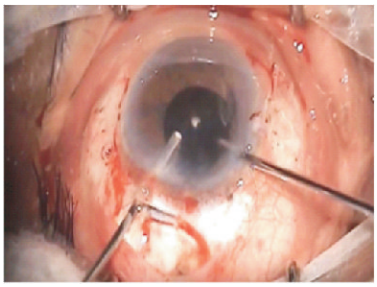


Figure 2 :

A 26G needle bent to 60° near the hub is inserted 1.5mm behind the limbus, near the 3 o clock scleral tunnel and brought out through the pupil and sclerocorneal wound.

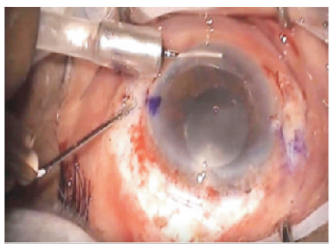


Figure 3:

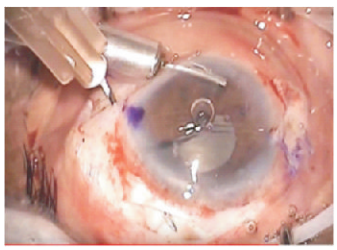
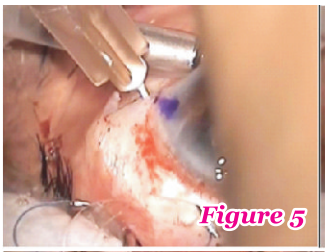


Figure 4:

4mm of leading haptic of a 3 piece IOL is inserted into the needle and the needle is exteriorsed through the sclerotomy.

The haptic is then tucked into the tunnel. Similar manoeuvre is performed near the 9 o clock tunnel and the trailing haptic exteriorised.



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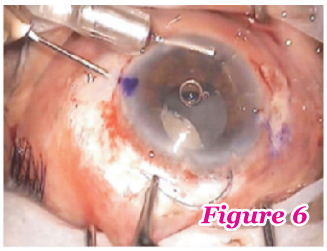
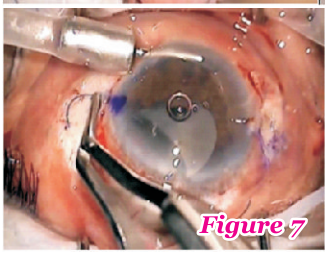


Figure 6



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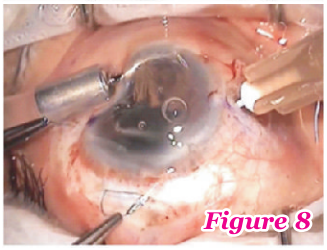

Figure 8

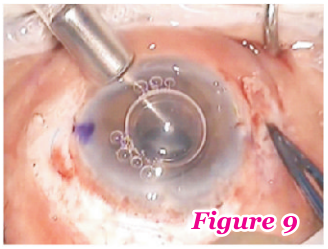

Figure 9

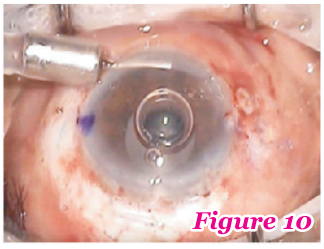

Figure 10

IOL centration is adjusted. AC maintainer is removed and conjunctiva closed.

Result: 5 cases have been performed by single surgeon (SM). At 6 month follow up of all patients, SFIOL was stable with improvement of visual acuity in all patients.

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Combined Mechanism Glaucoma

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Definition :

Eyes that have an occludable angle but fewer signs of angle closure not corroborating with the degree of raised intraocular pressure (IOP) and glaucomatous optic neuropathy are diagnosed as having 'combined mechanism' glaucoma (CMG). In other words, CMG is defined as a combination of both the primary types of glaucoma, the primary open angle glaucoma (POAG) and the primary angle closure glaucoma (PACG).[1,2]

Clinical Features :

The patients may present with non-specific symptoms, and it is more often detected incidentally, or as a part of screening programs.

- Age group: Commonly between 50-60 years of age
- Laterality: Usually bilateral.
- Baseline IOP: May vary from 23-28 mmHg.
- Anterior chamber depth: Usually normal centrally or slightly shallow, but may have narrow recess (Figure 1).

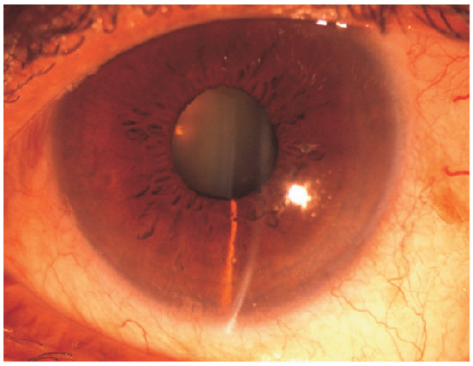

Figure 1 : Slit-lamp diffuse photograph showing a mildly shallow anterior chamber, patchy pupillary ruff atrophy and immature senile nuclear sclerosis.

- Gonioscopy: There would be an occludable angle (posterior trabecular meshwork not visible in at least 180°) which on manipulative/indentation gonioscopy show up limited signs of angle closure,i.e, goniosynechaie<90° and sparse blotchy pigmentation (Figure 2 & 3).

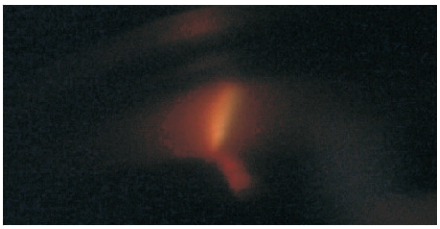

Figure 2 : Goniophotograph showing occludable angle

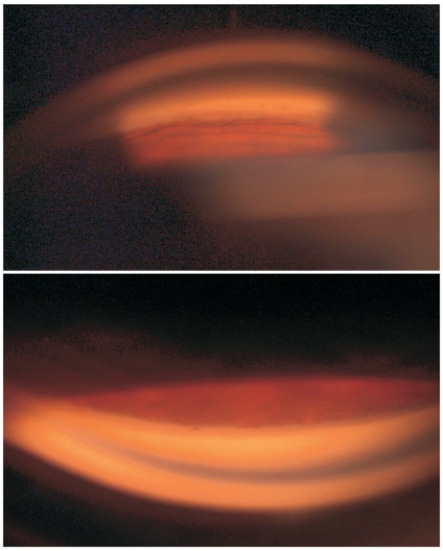

Figure-3 : On manipulation gonioscopy, the angle opens up to reveal the posterior trabecular meshwork and few goniosynechaie.

- Pupillary ruff:May show patchy ruff atrophy
- Iris pattern: May be decreased.
- Optic disc: Features consistent with glaucomatous optic neuropathy (Figure 4).

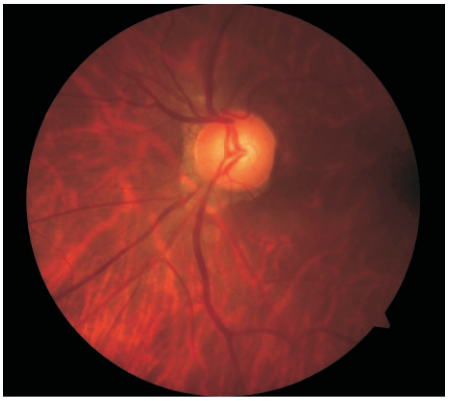

Figure 4 : Stereoscopic fundus photograph showing CDR 0.8:1

The sparse goniosynechiae and blotchy pigmentation imply that the patient has had fewer episodes of angle closure attacks in the past than of that which would be expected in a PACG eye with equal amount of optic neuropathy. Therefore, with the anterior segment picture not corroborating with the degree of optic neuropathy to either label a POAG or PACG, this conditionpresents as a combination of both of the primary types, and therefore referred to as the'combined mechanism glaucoma'.

Investigations

- Central corneal thickness (Pachymetry)
- Biometry (axial length, anterior chamber depth, lens thickness, white-to-white)
- Angle AS-OCT (angle opening distance, trabecular iris space area, angle recess area, trabecular iris angle, iridotrabecular contact)
- Visual field analyser
- Retinal nerve fibre layer OCT

Sihota et al[1] in their study on CMG patients showed that their mean corneal diameter was 12.11 ± 0.54 mm, axial length 23.48 ± 0.95 mm, anterior chamber depth was 3.06 ± 0.26 mm and lens thickness 4.44 ± 0.29 mm. On angle anterior segment optical coherence tomography (AS-OCT), the mean angle opening distance (AOD 500) was 0.32 mm and trabecular iris space area (TISA 500) was 0.13 mm^2. The mean circumferential iridotrabecular contact (ITC) in CMG eyes was 15% as against PACG whose mean ITC was 87% and POAG 0%. All these parameters fell in the mid-rangebetween those of POAG and PACG.

Differential Diagnosis :

The closest differential diagnosis to CMG is primary open angle glaucoma. A CMG could be missed if gonioscopy were not done in presumed POAG. It is important to differentiate CMG from the latter because the pathogenesis and management strategy differfor both. While POAG is primarily due to age related trabecular meshwork changes, CMG has anoverlap of the angle closure component.

Secondary glaucomas such as pseudophakic glaucoma, aphakic glaucoma, post-uveitic glaucoma and pseudoexfoliation glaucoma may have a normal central chamber depth and blotchy pigmentations and goniosynechaieon the angles. Hence, secondary glaucomas must be ruled out before making a diagnosis of CMG.

A confounder by name is the 'mixed mechanism glaucoma' which can be confused for CMG.[3,4] While CMG is a combination of the two primary glaucomas (POAG+PACG), mixed mechanism glaucoma is referred to when there is a combination of a primary and a secondary glaucoma (or)> 1 secondary glaucomas (eg, POAG + steroid induced glaucoma; post-uveitic + steroid induced glaucoma respectively).

The treatment of secondary and mixed glaucomasdiffer from primary glaucomas in that, they focus not only on the management of intraocular pressures, but also on the control of the inciting factor.

Treatment :

The management of combined mechanism glaucoma is distinctly different from that of POAG. In contrast to the latter, CMG eyes require a peripheral iridotomy in order to circumvent the existing relative pupillary block owing to their occludable angle status. This should be followed by medical management and measurement of diurnal variation of IOP four weeks later. Based on the highest readingrecorded, a target IOP should be set and treatment titrated accordingly.

Selective laser trabeculoplasty can be attempted in areas of visible trabecular meshwork, however results are not known at present.

In the event of failure to achieve target IOP or disease progressiondespite maximal therapy, filtering surgeries may be proceeded with.

Take home messages :

- Combined mechanism glaucoma isa distinct entity which is a combination of both the primary type of glaucomas, the POAG and the PACG.
- Eyes which have an occludable angle but goniosynechaie<90° not consistent with PACG fall into this category.
- Management includes a peripheral iridotomy followed by the medical/laser/surgical procedures as per the severity.
- Mixed mechanism glaucoma differs from CMG in being a combination of a primary and a secondary glaucoma (or)>1 secondary glaucomas.

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B-HEX® Pupil Expander - Pearls & Pitfalls

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The B-HEX® Pupil Expander (Med Invent Devices) is a disposable 6.5 mm flexible hexagonal device with notches at corners and flanges at sides.[1] Alternate flanges with holes are tucked under the iris to engage the notches to the margin of the pupil to provide a 5.5 mm expanded pupil. Unlike devices with scrolls or pockets which require an injector to avoid snagging the incision, the preloaded B-HEX is inserted and removed through a 1.5 mm or larger incision using a B-HEX® 23 gauge micro-forceps. The thin profile and uniplanar design allow unhindered instrument movement during phacoemulsification, cortical cleaning and IOL implantation. The B-HEX is safely used even after capsulorhexis since the thin uniplanar notches are directly visualized to avoid the capsule margin. It is useful in standard coaxial phacoemulsification, MICS, FLACS, small pupil pars plana vitrectomy and shallow anterior chamber eyes.

While the innovative and patented design of the B-HEX offers many advantages, there are a few limitations. The following tipswill help in safe and efficient usage of the B-HEX as well as other pupil expanders. Though the B-HEX is user friendly andrequires average skills, thesimple design is deceptive and has misled some surgeons to take the plunge without watching videos, resulting in unfavourable outcomes. Hence, it is strongly recommend that instruction videos are viewed at least before first use.

Keeping theanterior chamber (AC) under-filled with viscoelastic allows anteriorbowing of the Iris and helps in tucking of flanges. Over-filling viscoelastic in the AC pushes iris against lens capsule making tucking difficult. A little Viscoelastic injectedunder pupil margin to lift it off the anterior lens capsule further helps create space for flanges.

It is important to differentiate the 'Elastic'Intraoperative Floppy Iris Syndrome (IFIS) Pupil from the 'Rigid non-elastic'Pupil seen in pseudoexfoliation and uveitis. Pupil stretching is not effective in IFIS. The rigid pupiloften has a fibrous ringwith a fixed circumference and cannot be expandedlike an elastic band. This needs to be stretched by causing micro-tears of the pupillary sphincter.[2] A simple technique to differentiate between a rigid and elastic pupil is to inject BSS and inflate the AC after making the side-ports.

The elastic pupil would stretch a little and enlarge in size while the rigid pupil would not budge. On the one hand, abulky pupil expander will stretch the pupil but it is unwieldyand will also produce uncontrolled sphincter tears and on the other, the B-HEX is extremely thin and easy to handle but it does not have the mechanical strength to cause sphincter tears. With that trade-off, a pupil which is rigid and less than 4 mm, is better stretched bimanually to about 5 mm before placing Iris Hooks or a Pupil Expander. If a stretching is not performed it is very difficult to tuck the 3rd flange. The remedy is to either stretch the pupil with a partially engaged B-HEX or use a bimanual technique to tuck this flange. While the flange is held with the 23 g forceps, a Kuglen hook introduced through an opposite side-port retracts the pupil margin to facilitate bimanual tucking. Though all 3 flanges are engaged, since this rigid pupil still has a fixed circumference it will cause the B-HEX to buckle. Even at this stage a bimanual stretch of the 'B-HEX-Pupillary margin complex' will result in an expanded 5.5 mm regular hexagonal pupil.

The recommended standard technique is to hold the leading flange with the B-HEX 23 gauge forceps[3] and advance the B-HEX Pupil Expander into the AC till the entire device is in the AC and then place it on the Iris without any attempt to tuck the flanges. When the leading flange is held at the tips of the jaws of the forceps it is almost always possible to insert the entire device into the AC in a single pass. In smaller eyes, a part of the trailing flange may require to be tucked in in an additional step. The main and side-port incisions are to be used intelligently to access the flanges for tucking. By tucking the flange opposite the main incision first, the other two flanges to be tucked are very easily accessible using the side-port incisions. A flange may often be obscured by a dense Arcus Senilis or under incisions. A Kuglen hook can be used to nudge the partially engaged B-HEX to draw the obscured flange centrally and make it visible. If a large part or the entire B-HEX has inadvertently been placed behind the Iris, it is best

to retrieve the device and bring it entirely into the anterior chamber and restart the tucking process.

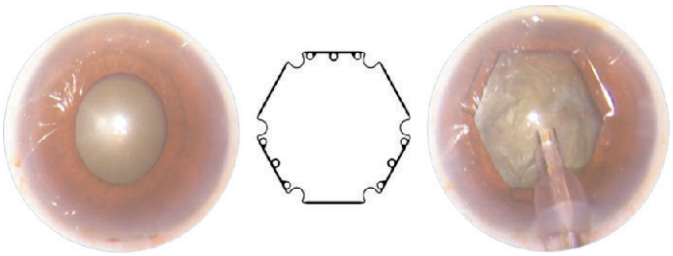

Figure 1: Alternate flanges of the B-HEX Pupil Expander tucked under the pupil margin to result in a hexagonal expanded pupil.

The B-HEX with its alternate flanges being anterior and posterior to the iris does dampen its billowing in an eye with IFIS. However, it must be realized that Iris hooks or pupil expanders do very little to prevent Iris prolapse in IFIS. Iris prolapse depends on various factors such as the extent of the IFIS pathology, AC depth,fluidics, incision construction, iris handling etc. What Iris hooks and Pupil expanders do is to provide an assured constant pupil size for uninterrupted visualization and safe phaco emulsification.

In a deep set eye, it is difficult to negotiate the side-port incision with the 23 g forcepsto gain access to the flanges of the B-HEX. It is useful to rotate the eyeto make theside-portseasily visible and accessible.When manipulations lead to large excursions rotating the globe outside the operating field, it is helpful to fixate of the globe and restrict its movement with a side port instrument.

The B-HEX is very safe for use even after the capsulorhexis. A little viscoelastic placed on the anterior capsular rim facilitates tucking of the flanges. The open notches are easily seen from the top view as the pupil margin is engaged. As a flange is tucked and carried a little distance to the periphery, if the capsulorhexis margin is visualized it is instant confirmation that the margin has not been engaged inadvertently.

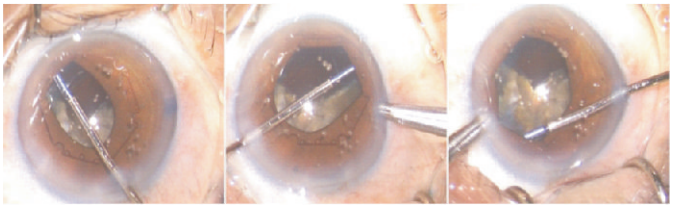

Figure 2: If the capsulorhexis margin is visualized it is instant confirmation that it has not been engaged inadvertently.

It is important to realize that the engagement of a pupil expander to the pupil margin is not very firm and it is very easy to dislodge it inadvertently. Since attempting to deliver a large nuclear fragment from inside the bag to the anterior chamber could result in such dislodgement, it is advisable to chop these fragments to smaller pieces. Again, care must be taken not to push the pupil expander with the leading or trailing haptic of the IOL. An IOL placed on the Iris can often be pushed into the capsular bag by forcing it against the pupil margin. However, this would not be applicable in the presence of a pupil expander because any attempt to push the IOL against the pupil margin will result in pushing the pupil expander and dislodging it either into the capsular bag or behind the iris. An attempt must be made to deliver the leading and trailing haptic in the bag in a single motion. If the trailing haptic is not delivered into the bag, there should be no attempt to push it against the pupil margin as this might dislodge the B-HEX. A Kuglen hook may be used to push orpull the outer edge of the trailing haptic in the IOL plane to cause flexion of the opposite (leading) haptic at the haptic-optic junction against the equator of the bag. This creates room for the trailing haptic to negotiatewithout contacting the pupil margin. In the rare event that the B-HEX is dislodged into the capsular bag or is entangled with an IOL in the bag, it can be easily cut with a pair of scissors and drawn out.

If there is nucleus drop and there is a need to refer the patient to a vitreo-retinal facility for secondary management, it is advisable to leave the B-HEX temporarily engaged and the pupil expanded so as to facilitate further management. The B-HEX is made of Polyimide which is used in IOL haptics and is a biocompatible material.

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Classification Of Pimary Angle Closure Disease

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Primary angle-closure disease (PACD) is the leading cause of irreversible blindness in East Asia and is responsible for half of the glaucoma related blindness.[1]

The classification of PACD is often considered confusing, due to inconsistencies in terminology and nomenclature various described and recently used classifications are following.

Classification of PACD :

Angle closure or narrow angles are the essential components of PACD, while elevated intraocular pressure (IOP) is secondary to angle closure. PACD can be classified in different ways, based on clinical presentation, natural history, anatomy, etiology, etc.

Clinical classification/ Clinical Spectrum of PACD :

It is based on the clinical course of primary angle closure glaucoma (PACG) and therefore, it revolves around the course of the disease and time of onset.[2]

a) *Latent PACG:* These are the eyes where pigmented trabecular meshwork is not visible in more than 2 quadrants without indentation or manipulative gonioscopy. There are no other symptoms or signs like any evidence of gonioscopic abnormalities and raised IOP.

b) *Subacute / Intermittent PACG:* These are the eyes with sudden closure of entire angle for a short period of time under some physiological factors like reading in dim light, entering into a darkened room etc. but spontaneous resolution ofpupillary block is the rule. Therefore, these have prodromal symptoms of mild headache, blurred vision, colored haloes that resolve spontaneously. Anterior segment may show subtle signs of angle closure like pupillary ruff atrophy, iris atrophic patches etc. and Gonioscopic abnormalities like patchy pigmentation/ synechia might be present but with normal IOP in interparoxysmal phase.[2]

c) *Acute PACG:* In this stage the sudden complete closure of entire angle leads to sudden rise of IOP to very high levels. This results in the sudden onset of classical symptoms of redness, pain, watering, photophobia, colored haloes and classical signs of circumciliary congestion, corneal edema, mid dilated pupil, glaukomaflecken etc.

d) *Chronic PACG:* As the name suggests, the closure of trabecular meshwork by the iris is slow and steady. This may be due to creeping angle closure or following acute PACG or due to recurrent subacute attacks. These eyes have chronically raised IOP with PAS in more than two quadrants. As these eyes have enough time to accommodate for raised IOP, therefore the symptoms are not at all dramatic.

The main concern in this classification is that at certain occasions subjects with "latent angle closure glaucoma" have been classified as cases of established glaucoma, despite having normal visual function. This resulted into misinterpretation of the estimates of visual morbidity attributable to glaucoma.[3] Moreover, difference in the definition of PACG and POAG made it difficult to compare the prevalence and study risk factors in epidemiological glaucoma research.[4]

Pathogenic classification:

This classification is based on anatomical levels of obstruction to aqueous flow in primary and secondary angle closure glaucoma.[5] According to this the angle closure may be due to forces acting at four anatomical levels:

a) *Level I:* The forces acting at the level of iris that includes-
- pupillary block
- non-pupillary block
- angle crowding mechanisms

b) *Level II:* The forces acting at the level of ciliary body, including plateau iris configuration and iridociliary cysts.

c) *Level III:* The forces acting at the level of lens, including thick, anteriorly positioned and subluxated lens.

d) *Level IV:* The vector forces posterior to lens – that includes aqueous misdirection, choroidal effusion, space-occupying lesions etc.

However, clinical application of this classification is difficult at various occasions as in many ACG patients multiple "levels" may simultaneously or consecutively play a role. Moreover, estimate of visual morbidity cannot be correlated.

Thus, it was thought that a full re-evaluation of the definition is mandatory, with emphasis on visual morbidity

rather than symptomatic disease or pathogenic mechanism.

Hence, At the biennial congress of the International Society for Geographical and Epidemiological Ophthalmology (ISGEO) held atLeeuwenhorst, the Netherlands, in June 1998, a group interested in glaucoma epidemiology met to propose the new Epidemiological classificationbased on progression of the disease.[4]The recent American Academy of Ophthalmology (AAO) guidelines also follows the similar classification. [3]

Epidemiological classification

The fundamental concept of this classification is that the term 'glaucoma' is reserved for people with established, visually significant and with end organ damage. [4] So, this classification can also be used conveniently for cross sectional epidemiological research.

(a) *Primary angle closure suspect (PACS):* PACS was defined as an eye with occludable angle (pigmented trabecular meshwork not visible for ≥ 180° under static gonioscopy without peripheral anterior synechiae, PAS) and IOP lower than 21 mmHg, and no glaucomatous optic neuropathy (GON).

(b) *Primary angle closure* (*PAC*): PAC is defined as an eye with an occludable angle and gonioscopic features indicating that trabecular obstruction by the peripheral iris has occurred (e.g. iris whorling, PAS, "glaucomflecken", lens opacities or excessive pigment deposition on the trabecular surface) or raised IOP (>21 mm Hg) but the optic disc does not have GON.

Thus, in this new concept, PAC includes both asymptomatic people with occludable angles who either have not or have had an acute attack, that was treated promptly but suffered no detectable GON damage.

(c) *PACG*: These were the eyes with PAC and GON (defined as a vertical cup/disc (C/D) ratio >0.7 and/or C/D asymmetry >0.2 and/or focal notching), with compatible visual field loss on static automated perimetry.

This classification is not intended to indicate that those with PAC do not require treatment. It is intended to differentiate between those with and without damaged visual function attributable to GON. Moreover, it is seen that both are likely to benefit from iridotomy, but the former (PAC) are likely to be cured, while the later will require more intensive follow up and treatment much like the treatment for POAG.[2,4]

To summarize, the epidemiological classification has been accepted universally and is used to classify PACD. It differentiates well between those with and without visual morbidity and therefore assists in defining the treatment protocol.

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JOURNAL UPDATE

Novel Cyclosporine A Formulation shows promise in patients with Vernal Keratoconjunctivitis

Investigators of this phase 3 trial assessed the efficacy and safety of an investigational cationic emulsion Cyclosporine A (CsA CE) for severe vernal keratoconjunctivitis. They randomized 169 pediatric patients to a high or low dose of CsA CE drops or a vehicle control. Both treatment groups showed significant improvements over the vehicle group in corneal fluorescein staining scores and rescue Dexamethasone use. Additionally, the high-dose arm reported improved keratoconjunctivitis symptoms and quality of life compared with vehicle. The novel formulation was well tolerated with no unexpected safety findings. Ophthalmology, May 2019

Pterygium Auto Graft : Inferior Found To Be Superior

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Abstract

Topic: Pterygium auto graft: Inferior found to be superior

Introduction: Pterygium is an elastotic degeneration of sub epithelial conjunctival tissue which encroach the cornea. Surgical excision is preferred method of treatment. Conjunctival autograft has shown promising results with low recurrence rate. The superior-temporal conjunctiva is mostly used for this purpose but this leads to non availability of superior conjunctiva for future surgical intervention. This study was conducted whether the inferior conjunctival graft functioned similar to the superior graft and whether there was any difference in recurrence of pterygium in between a superior and inferior conjunctival graft.

Methods: A total of 40 patients with primary pterygium in age group of 20-50 years with >2mm of horizontal extension without any ocular and systemic disease were included in the study. All patients were divided into 2 groups of 20 each. Group A received superior graft while Group B received inferior grafts. Patients were followed up on day 1, one month, 3 months and 6 months.

Results: A total of 40 patients (20 in each group) were participating in the study, out of which 32 male and 8 female patients. In group A, 1 patient at 3 months and 3 patients at 6 months have recurrence of pterygium while in group B, only 2 patients show recurrence at 6 months follow up.

Conclusion: This study concludes that the recurrence rate is less with the inferior conjunctival grafts. It also shows that inferior conjunctiva is easily assessable and had less post operative symptoms of foreign body sensation.

Introduction

Pterygium, derived from the ancient Greek word "pterygos", denoting a wing. This is a degenerative condition of conjunctival tissue. This is a common ocular surface lesion originating in the limbal conjunctiva within the palpebral fissure with progressive involvement of the cornea. It proliferates as a vascularised granulation tissue invading the cornea and destroying the superficial layers of the stroma and Bowman's membrane.

The prevalence of pterygium varies from 1 to 15% depending on the geographical location of the population. The main risk factors are the total exposure to ultra-violet (UV) light and increasing age. One most probable hypothesis for its pathogenesis is that the effects of UV radiation cause actinic changes in the conjunctiva, resulting in abnormal growth. Actinic changes seen on histopathology similar to actinic keratoses on the skin also supports the role of UV radiation. This growth may be exacerbated by hot, dry or windy environmental conditions. Those patients with lifestyles that have greater exposure to these conditions e.g. outdoor activities or farmers or labourers; have a greater risk of developing pterygium. Recurrent pterygia appear to be more related to surgical trauma than UV radiation as avoidance of UV radiation has not been shown to affect the incidence of recurrence.

Patients with pterygium present with various complaints, ranging from no symptoms to significant redness, swelling, itching, irritation, and blurring of vision associated with elevated lesions of the conjunctiva and contiguous cornea in one or both eyes. The diagnosis of pterygium is based on the clinical appearance of the lesion. Typical findings include triangular shaped fibrovascular conjunctival growth within the palpebral fissure which may be extending onto the corneal surface. A pigmented epithelial iron line (Stocker's line) adjacent to a pterygium is evidence of chronicity. Pterygium can be graded as:

- **Grade I** covered pterygium that was between the limbus and a point midway between the limbus and the pupillary margin.
- **Grade II** occurred when the head of the pterygium was present between a point midway between the limbus and the pupillary margin & the pupillary margin
- **Grade III** covered pterygium that crossed pupillary margin.

There is no medical management and surgical removal is the preferred method of treatment. Conjunctival autograft with limbal stem cell transplantation has shown promising results with low recurrence rate. Conjunctival autograft is usually obtained from the supero-temporal area which may adversely affect the outcome of future filtration surgery. If supero-temporal area is kept untouched the success of glaucoma

surgery can be helped. Keeping the above facts in mind, we retrospectively compared success rate of the superior and inferior conjunctival grafts.

Materials & Methods

This was a Prospective interventional studyconducted over a period of 6 months from November 2017 to June 2018 in a tertiary eye care center. This study was conducted in 40 eyes which were divided into 2 groups with 20 eyes in each group.

- Group A of 20 patients treated by pterygium excision along with the superior temporal bulbar conjunctivae (STBC) autograft.
- Group B of 20 patients treated by pterygium excision along with the inferior temporal bulbar conjunctivae (ITBC) autograft.

Inclusion criteria :

- All patients 20-50 years of age group who had progressive pterygium and are willing for surgery
- Ready to give informed consent for the study

Exclusion criteria :

- Patients with trichiasis, entropion, ectropion
- Lacrimal aparatus active disease
- Recurrent pterygium
- Systemic illness like Diabetes

All patients reporting to the hospital with primary pterygium in the age group of 20-50 years were included in the study after obtaining informed consent. A comprehensive evaluation was undertaken including patient's age, gender, medical and ocular history, visual acuity assessment and slit lamp examination. All surgeries were carried out by single surgeon under local anesthesia. The pterygium was dissected using a blade. Subconjunctival fibrous tissue under pterygium was excised wider than the pterygium size. A free conjunctival limbal auto graft was placed over the bare sclera with help of serum. Postoperative visit on day 1, 1 week, 1 month, 3 months and 6 months from the date of surgery. Data was recorded in self made detailed proforma. Data analysis was done by Statistical Package for Social Sciences (SPSS) version 21.0 statistical analysis software.

Results and Observations

- A total of 40 patients (20 in each group) were participating in the study, out of which 32 male and 8 female patients.

Group	Male	Female
A	15	5
B	17	3

- When we classify them according to grading of pterygium the maximum number of patients fall in the grade II pterygium.

	Grade I	Grade II	Grade III
Group A	6	13	1
Group B	7	11	2

- 2 patients in each group did not complete the follow up so we excluded them from further calculation.
- Post op vision: 12 patients in group A and 10 patients in group B appreciated vision improvement on Snellen's chart while 6 patients in group A and 8 patients in group B reported no change in visual status.
- During follow up period post-operative self limiting complications occurred in the first week in 4 (22.22%) cases in group A while 3 (16.67%) cases in group B as foreign body sensation, photophobia and blepharospasm.
- **Recurrence:**
  - In group A, 1 (5.56%) patient at 3 months and 3 (16.67%) patients at 6 months have recurrence of pterygium of grade II.
  - While in group B, only 2 (11.11%) patients show recurrence of pterygium of grade II at 6 months follow up.

	Group A	Group B
Day 1	0	0
1 Week	0	0
1 Month	0	0
3 Month	1	0
6 Month	3	2

Discussion

Various surgical techniques have been used to treat pterygium. The diversity of techniques reflects the ongoing surgical challenge to devise the best method for treating pterygium. Many studies have been published with conflicting results. In our study we compared and evaluate the success rates of pterygium excision with conjunctival autograft transplantation with graft from the superior temporal bulbar conjunctiva (STBC) and pterygium excision with conjunctival autograft transplantation with graft from the inferior temporal bulbar conjunctiva (ITBC).

In our study, most patients were male. This

predominance in this study can be explained by work related sun and heat exposure in males. Vision improvement in both groups can be explained by the pre-op grading of pterygium and it has no role with the group allotted. After assessing the self limiting complications like foreign body sensation, photophobia and blepharospasm it was found that these problems were more in group A patients then in group B and these problems were significantly reduced after second and third follow ups. In group A, 3 patients while in group B, only 2 patients show recurrence at 6 months follow up. So less in group B.

In other studies Koc et al demonstrated that autografting from superior or inferior sites showed no significant difference in recurrence rate while Nazzulah et al and Yeung et al, reported low risk of recurrence following inferior temporal conjunctival autografting.

Limitations of study

- We used convenient method of sampling and all patient were taken from the charitable hospital
- Most of patients are of low socioeconomic status and this will affect compliance which ultimately affect the recurrence rate
- Randomisation was not done
- Sample size was small
- Poor post operative follow up

Conclusion

This study concludes that pterygium excision with conjunctival auto-graft transplantation from the inferior bulbar conjunctiva is highly efficient in terms of post op patient symptoms, safety, efficacy, easy assessability and low recurrence rates. The low recurrence rate of the inferior limbal conjunctival auto graft and the few self limiting complications makes this procedure of choice in primary pterygium, especially in glaucoma patients in whom the superior bulbar conjunctiva is valuable.

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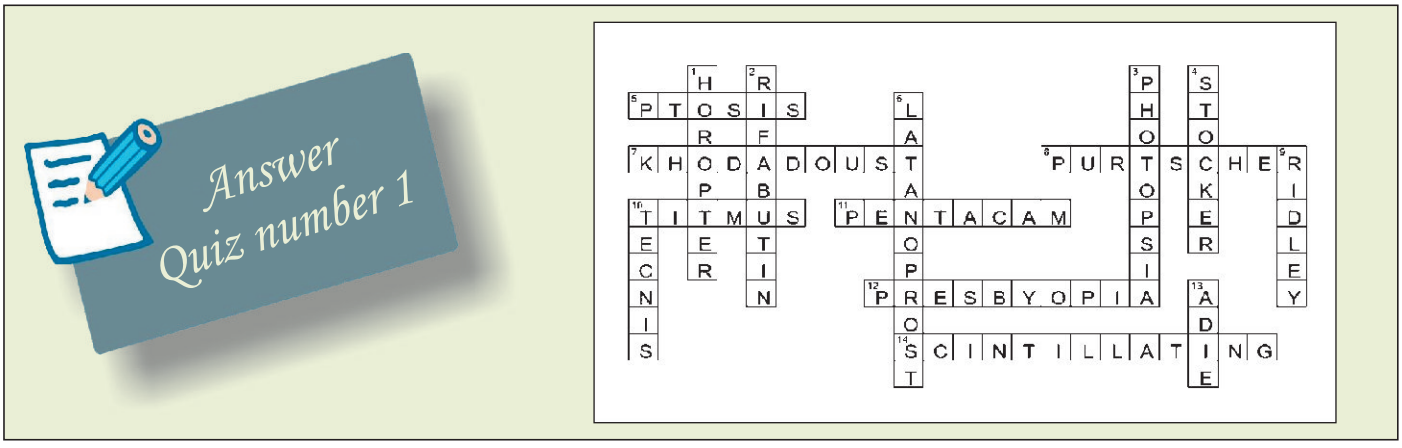


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Orbital Cellulitis- A Review

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Abstract :

Orbital cellulitis is an infective process involving ocular adnexal structures posterior to the orbital septum.[1] It is an ocular emergency that not only threatens vision but also can lead to life-threatening complications such as cavernous sinus thrombosis, meningitis, and brain abscess. Visual loss ranges from 7.1% to 23.6%, which may be due to optic atrophy, central retinal artery occlusion, or exposure keratopathy with ulcer formation.[2,3] Catastrophic local sequelae (infarction of optic nerve, sclera, choroid, retina) and intracranial spread (in sinus infections most common being frontal sinus, followed by ethmoid and maxillary sinuses) occur in untreated cases.The prognosis is good if prompt medical treatment is received.

Orbital cellulitis is an infective process involving ocular adnexal structures posterior to the orbital septum.[1]

Source of infection :

Infection into the orbit may spread through direct extension from the paranasal sinuses and other periorbital structure, or direct inoculation of the orbit from trauma or surgery, or through hematogenous spread (bacteremia). Most commoninfective organism isStaphylococcusaureus in adults, and Streptococcus speciesin pediatric population.[4]

Access routes :

Infection can gain access into the orbit through thin bones of the orbital wall, venous channels, foramina and dehiscences. Medial orbital wall is thin and perforated bymanyvalveless blood vessels, nerves and numerous defects (laminapapyracea/Zuckerkandl dehiscences).[5]

Infections may also spread as direct extensions from ipsilateral ethmoid or frontal sinuses, and indirectly from maxillary sinus, secondary to dental infections. It can be caused by microorganisms indigenous to the mouth, including anaerobes, commonly Bacteroides species.

Extension through soft tissue routes occurs posteriorly in the orbit where the fascia between the rectus muscles is thin & often incomplete. This allows easy extension between the extraconal and intraconal orbital spaces.[6] Also, access can occur through venous route. Orbital veins are valveless which allow passage of infection, both anterograde and retrograde.

Pathophysiology :

Edema of the sinus mucosa results in narrowing of the sinus ostia causing cessation of normal sinus drainage. Subsequently, proliferation of microflora occurs which invade the edematous mucosaresulting in suppuration. Suppurative process is enhanced by the reduced oxygen tension within the obstructed sinus cavity.

Epidemiology and Incidence :

The incidence increases during winter season in western countries whereas in India it increases in monsoon.

The frequency of orbital complications from sinus infection ranges from 0.5% to 3.9%.Ipsilateral ethmoidal disease is usually present, frequently encountered with ipsilateral maxillary sinus infection.[3] Frontal sinus involvement is frequently encountered in adolescents and adults. [2,7,8]

The incidence of orbital or periorbital abscesses varies considerably from 0% to 25% in different studies.[9]

Chandler's classification[5]

- Group 1 Pre septal cellulitis
- Group 2 Orbital/post septal cellulitis chemosis
- Group 3 Sub periosteal abscess
- Group 4 Orbital abscess
- Group 5 Cavernous sinus thrombosis

Pre-septal cellulitis :

Preseptal cellulitis describes an infection of the eyelid and superficial periorbital soft tissues (Figure 1) without the involvement of the globe and orbit. It occurs more commonly than orbital cellulitis and is generally associated with a more favorable prognosis.[10]

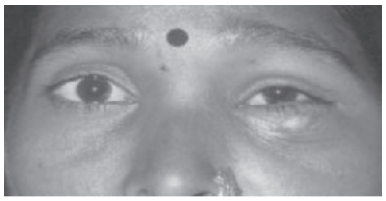

Figure 1: Clinical presentation of preseptal cellulitis (left side)- eyelid edema and erythema, features characteristic of cellulitis.

It presents with eyelid edema and erythema,with normal vision, absence of proptosis and chemosis, and full ocular motility without pain on movement.

On computerized tomography (CT) scan, diffuse soft-tissue thickening and areas of contrast enhancement anterior to the orbital septum are seen inpreseptal cellulitis (Figure 2 and 3).

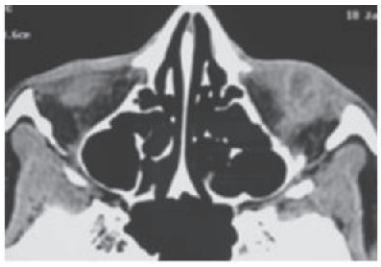

Figure 2 : CT scan (axial section) - diffuse contrast enhancing soft tissue thickening anterior to the orbital septum (left side)

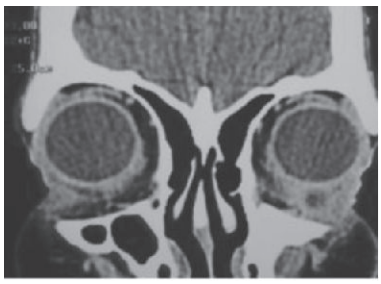

Figure 3 : CT scan (coronal section) - diffuse contrast enhancing soft tissue thickening anterior to the orbital septum (left side)

Subperiosteal abscess :

It results from progression or spread of orbital cellulitis beneath the periosteum of ethmoid, frontal or maxillary bones.

CT Scan-Low density collection beneath the periostem, adjacent to a sinus (Figure 4).

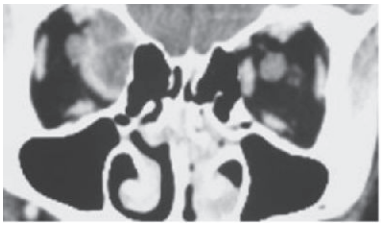

Figure 4 : CT scan (coronal view) showing a superior/ medial orbital subperiosteal abscess on the right side

Orbital cellulitis :

Orbital cellulitis involves infection of tissues posterior to the orbital septum, within the bony orbit (Figure 5). It is more common in the pediatric population. In contrast to the more common preseptal cellulitis, orbital cellulitis may be associated with significant complications, and thus, prompt diagnosis and treatment are important.

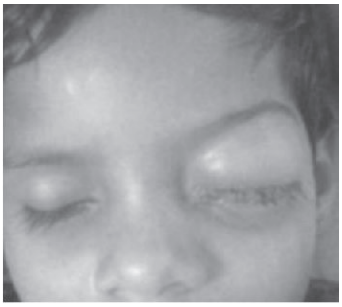

Figure 5 : Clinical picture of orbital cellulitis – proptosis with eyelid erythema and edema (left side)

Risk factors :

- Diabetes mellitus (especially those with diabetic ketoacidosis)
- Multiple blood transfusions
- Immunocompromised patients with severe neutropenia (steroid, non-steroid,disease)

Ocular Signs:

- Eyelid edema and erythema
- Conjunctival chemosis
- Proptosis
- painful ophthalmoplegia
- Decreased vision
- Relative afferent pupillary defect
- Resistance to retropulsion of the globe
- Elevated intraocular pressure
- Vision may be normal early, but it may become difficult to evaluate in very ill children with marked edema.

Systemic Signs :

- Fever
- Headache
- Rhinorrhea
- Malaise

Coexistent conditions :

- In the pediatric group- In more than 91% cases, radiologically confirmed ipsilateral sinus disease is present. The involvement of ethmoid sinus (43% to 75%) is more than maxillary sinuses
- In adolescents and adults, frontal sinus disease is more common.

Laboratory Studies :

- Complete blood count (CBC). Leukocytosis>15,000 with a shift to the left is present.
- Blood cultures are done prior to starting antibiotic treatment.
- Purulent material assessment -Gram stain and Culture in

both aerobic & anaerobic media

Commonly reported bacteria :

- Staphylococcus aureus (most common)
- Staphylococcus epidermidis
- Streptococci(paediatric age group)
- Diphtheroids
- Haemophilus influenza
- Escherichia coli
- No growth in up to 25% of orbital abscesses

Radiology :

- Ultrasonography - in-office screening procedure in cases of suspected orbital abscesses,shows low internal reflectivity. It detects abscesses of anterior orbit or medial wall with 90% efficiency.[11] Acute abscess may be poorly delineated.
- Computerized tomography scan- It is the investigative procedure of choice to diagnose orbital infection. Ct scan is necessary to assess orbit & sinuses and presence of any intracranial extension. It is also indicated in cases not responding to intravenous antibiotic therapy even after 24-48 hours. Plain and contrast CT (axial, coronal and sagittal) with thin sections (2mm cuts) are ideal.

CT-scan Signs :

- localized, generally homogenous elevation of the periorbita
- loss of delineation of tissue planes (Figures 6 and 7)
- Abscess -thick hyperdense wall with central hypodensity. (Figure 8)
- Thickening of ocular coats and Optic Nerve.
- opacified sinuses (ipsilateral)
- X-ray- Air-fluid level present, in an abscess cavity. However, gas-free abscesses may not be readily visible.[6]

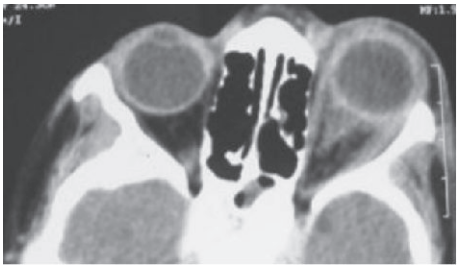

Figure 6 : CT scan (axial view) showing multiple loss of tissue planes in the left orbit.

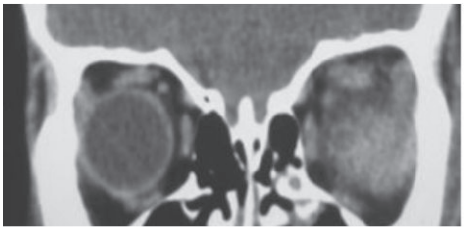

Figure 7 : CT scan (coronal view) showing diffuse inflammation in the intraconal and extraconal space of the left orbit.

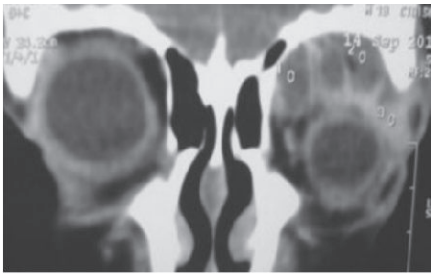

Figure 8 : CT scan (coronal view) showing multiple thick walled cavities (abscesses) in the left superior orbit.

Treatment :

- Medical Therapy-Hospital based
- Antibiotic therapy: emperical intravenous broad spectrum antibiotics. Oral antibiotics are givenon discharge.
- Anti-inflammatory: NSAID +/- Corticosterid
- Supportive treatment in form of supplementation of multivitamins, treatment of sinusitis and concurrent systemic illnesses.

Inter-speciality Consultation :

- Otorhinolaryngology consultation
- Other specialists – paediatrician, infectious disease specialist and radiologist.
- Neurosurgical consultation is indicated if brain abscesses appear

Surgical therapy for orbital abscesses :

- Surgical procedures- drainage of abscesses, canthotomy and cantholysis in orbital compartment syndrome.
- Influenced by visual status, size and location of orbital abscess and associated intracranial complications.
- Harris and Garcia recommendationsfor surgery in orbital cellulitis: presence of compromised optic nerve or retinal function, large abscesses and intracranial complications. [12]

Indications for Drainage :

- Poor response to appropriate antibiotic therapy within 24-48 hours
- CT scan shows opacified sinuses
- Intraorbital abscess / subperiosteal abscess, especially in an adult.

An expectant approach :

- Patients younger than 9 years of age in whom simple infections are suspected. Surgery may be warranted if:
- There is no clinical improvement in a timely manner
- Telative afferent pupillary defect develops at any time
- Fever does not abate within 36 hr
- Deterioration despite 48 h

- Improvement of CT findings lag behind the clinical picture

Follow-up :

Patients with severe orbital cellulitis often follow a protracted course. Repeat surgery may be required.

Patients are ideally monitored by an ophthalmologist, ENT specialist, and infectious disease specialist until symptoms, fever, WBC count, and imaging confirm that antibiotics can be discontinued.

Complications :

- Cavernous sinus thrombosis - bilateral disease with ophthalmoplegia and loss of vision.[13]
- Imaging studies are indicated when neurologic signs are present, to rule out associated epidural or subdural empyema, brain abscess, or cavernous sinus. [10,13,14]

Loss of Vision :

Mechanism – exposure keratopathy, optic neuritis, ischemia from thrombophlebitis and compressive central retinal artery occlusion.[15,16]

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Cataract surgery appears safe for patients with cancer

This is the first matched population-based analysis to assess the risk of endophthalmitis in patients with cancer. Researchers identified 23,362 Taiwanese adults who underwent bilateral cataract surgery at an average of 4.3 years after a cancer diagnosis. The rate of endophthalmitis during the 3-month follow-up period was similar between the cancer and matched noncancer groups (0.24% vs. 0.23%; P=0.892). Given these findings, the authors suggest cataract surgery can be performed in cancer patients when indicated. American Journal of Ophthalmology, March 2019

Diurnal Variation of IOP: The Triggerfish CLS

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Intraocular pressure is the most commonly identified risk factor and the only one which can be modified, in patients of glaucoma. Decrease in IOP has shown to be effective in retarding the progression of glaucoma, over a wide spectrum of disease, regardless of baseline IOPs. In addition, there is considerable evidence that IOP variables including peak levels and fluctuations (both short term and over longer periods) adversely impact the disease development and progression, acting as independent risk factors.

Why is 24 hour IOP monitoring essential?

Circadian fluctuation of IOP is known to be one the major risk factor for visual field progression in both PACG and POAG patients.IOP spikes have also been related to progressive visual field loss. Moreover, increased diurnal variation in IOP is an independent risk factor for glaucoma progression.[1]

According to various studies, glaucomatous progression occurs despite adequately controlled IOP measured in the office.

Various investigators have concluded that only office hours IOP recordings can be fallacious and are not enough for the management of glaucoma. Peak IOP occur outside office hours in almost half of our patients, with as many as 35% patients undergoing an immediate treatment change, following 24 hour IOP monitoring.

What are the options for 24 hour IOP monitoring?

A. Hospitalisation for IOP

The most important consideration for this is issues of cost and access. Moreover, one must ensure that the same tonometer is used in both sitting and supine positions. Therefore, options include theTonopen, Pneumatonometer, and Perkins.

Also, the same person must do the entire diurnal variation curve, to remove inter-observer bias.

B. Patient self-tonometry

This is fraught with questions about reliability of the readings. Also, since the patient wakes up, and sits up to take the readings, they are not really physiological records of IOP. Home tonometers which may be used include the I-care one, Zeimers, and Proview.

C. Sleep laboratory

Sleep labs provide the best analog for physiological IOP curves, however, they are expensive, and few.

What is currently the best bet for 24 hour IOP monitoring?

The triggerfish contact lens sensor (Sensimed, Switzerland) is a disposable silicon contact lens with an embedded micro electrical system,[2] which measures changes in corneal curvature induced by variation in IOP (Figure 1). In use in Europe with a CE mark for almost a decade, this device has recently received approval by the US FDA as well.

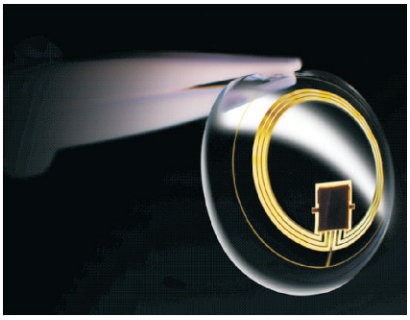

Figure 1: Triggerfish CLS sensor for 24 hour IOP monitoring

The Triggerfish is a silicon soft contact lens 14.1 mm in diameter and 585 µm in thickness in center. The device has 3 base curves (8.4, 8.7 and 9 mm) for a better fit, depending on the patient's corneal curvature (Figure 2). Embedded within the center are two strain gauges, a microprocessor and an antenna. [3,4]

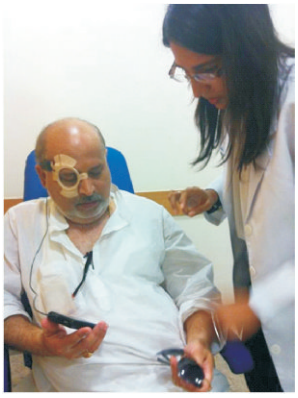

Figure 2: Patient wearing the Triggerfish CLS sensor for 24 hour IOP monitoring

These strain gauges detect changes in corneal shape, which correlates the CLS output and imposed IOP. The contact lens receives power from and transmits strain gauge information to an adhesive antenna that is pasted around the eye of the patient.

This antenna sends information to the portable recorder, which is worn by the patients around their waist, much like the holter used for ambulatory ECG recording.

What is remarkable is that the Triggerfish CLS takes 300 readings over 30 second period every 5 minutes (Figure 3). [5] This means that a total of 86,400 data points corresponding to IOP are recorded over a 24 hour period. This data is sent via blue tooth to a computer for analysis. The data points are measured in millivolts or millivolt equivalent, and are known to correspond to IOP fluctuations.[6,7]

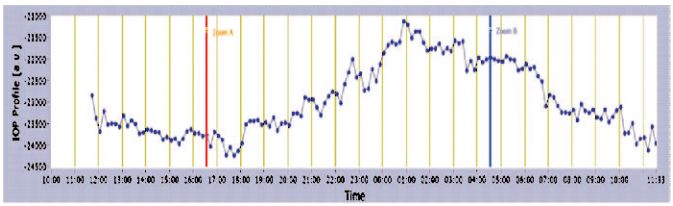

Figure 3: Diurnal variation in IOP as recorded by Triggerfish CLS sensor during 24 hour IOP monitoring

What is the current status of the Triggerfish CLS

The biggest advantage of the CLS is that the patient can be ambulatory during the measurements. The recordings therefore are true physiological IOP levels during the patients' daily activities. The patient is also encouraged to keep an activity log so that changes in IOP can be correlated with the activities at that time. Therefore appropriate feedback can be provided especially in regard to lifestyle modification. [3]

Various clinicians and researchers have found that the device is well accepted and tolerated by patients, with few complications. The CLS has been used to determine both diurnal variation in eyes of PACG and POAG, as well as to ascertain the effects of glaucoma therapy, both topical and following laser treatment.

The biggest disadvantage of the CLS is that the readings are in milli volt equivalent rather than mmHg. The recordings therefore cannot be interpreted as IOP in the clinical situation, since the relationship between the two parameters is nonlinear and influenced by viscoelastic properties of eye. Its other disadvantage is its high cost, since the device sensor is a disposable CL.

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JOURNAL UPDATE

Tocilizumab benefits patients with refractory CME

Researchers retrospectively assessed the effectiveness of tocilizumab in 25 patients with persistent cystoid macular edema (CME) secondary to noninfectious uveitis. Twelve months of tocilizumab therapy significantly improved macular thickness and BCVA and cut the mean dose of prednisone from 15.9 to 3.1 mg/day. Fourteen patients achieved remission and only minor side effects were noted. The authors conclude that patients treated with tocilizumab generally experience a quick and maintained response. American Journal of Ophthalmology, April 2019

Peri-Orbital Necrotising Fascitis

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Necrotising fasciitis is a life threatening infection involving fascia and necrosis of subcutaneous tissue. Peri orbital necrotizing infection has a better prognosis than that of necrotizing fasciitis of any other part of body. Early diagnosis and debridement is the treatment of choice. Here we report two cases of peri-orbital necrotizing fasciitis.

Key Words: Peri-orbital, Necrotising fasciitis, Debridement

Introduction

Necrotising fasciitis is a life threatening infection where skin and subcutaneous tissue gets affected. It is rapidly spreading disease where necrosis extends along superficial fascial plane. Generally NF develops in area of compromised skin integrity. The portal of entry usually is a trivial trauma or surgical wound. However ,no definitive cause can be found in upto 30-50% of cases.[1-3] Few co-morbid conditions are found associated with necrotizing fasciitis like Diabetes Melitus (31-44%), Obesity (28%), Smoking (27%), Alcoholic abusers (17%),Cirrhosis(8-15%), Malignancy(3%), Corticosteroid therapy(3%),and Chronic renal failure(3%).[4-5] The documented incidence of NF in literature is reported to be 0.4 cases per 100,000 in UK, but Indian scenario is not clear owing to paucity of literature in our setting.[6]

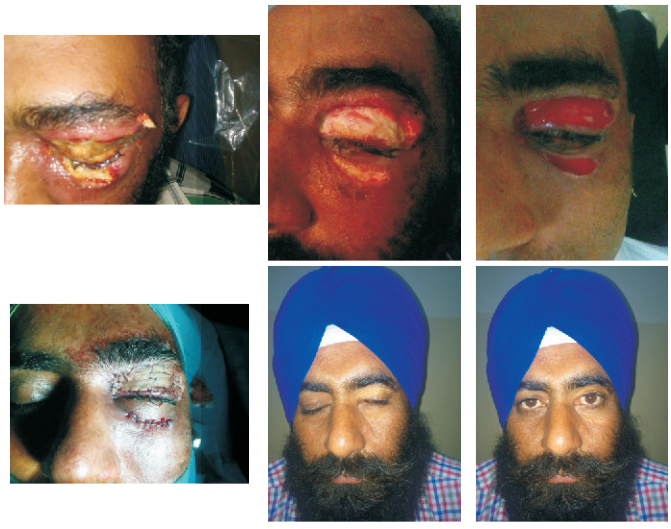
Morbidity and mortality of NF is very high but it vary according to area involved. Per orbital necrotizing fasciitis is very rare with incidence reported in UK .24 per 1,000,000 per year.[7] It is generally thought to be the least severe form of NF with best prognosis.[8]

Case Report

Case 1 A 45 yr old male, chronic alcoholic presented to us with a history of fall after having alcohol. During fall he got hit by glass table over face. The patient developed redness and swelling over left cheek, eye (peri orbital area) after 6 hrs . Patient didn't take any treatment for that. Three days after the fall, patient developed gangrene of peri orbital skin. He came to our OPD fourth day after fall. On examination he was febrile. His both upper and lower eyelids were markedly swollen and there was slough over both the lids. Edema and erythema was extending over to the left cheek. At that time eye examination was not possible as he was not able to open eye but there was no proptosis.

We sent the lab investigations of the patient,took the patient to operation theatre and debrided the gangrenous skin and subcutaneous tissue ,removed the slough.Patint was put on IV antibiotics Inj Piperacillin and Tazobactum,Inj Amikacin and daily dressings wee done. Repeated debridements were done as and when required. Wound swab was sent for culture and sensitivity. Slowly swelling started decreasing and granulation started appearing. When swelling subsided completely we did complete eye examination. His visual acuity was normal(6/6) and ocular movements were normal. Lid margins and lashes were spared.

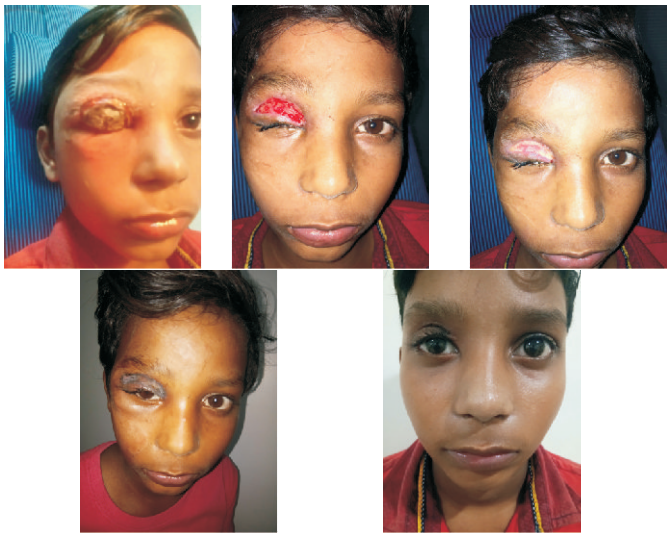
It took almost 15 days for healthy granulation tissue to appear. When culture report showed no organism and there was healthy granulation tissue we took the patient for reconstruction. Under GA taking all aseptic precautions split skin graft was harvested from his left arm medial aspect and used to cover the defect of both upper and lower eyelids.Tie over dressing done and IV antibiotics continued. Postoperative period was uneventful. Graft uptake was 100% and patient was now able to open and close the eye and was highly satisfied. ***(Figure 1-6)***



Case 2 7yr old male child presented to our OPD after a trivial trauma to face at school. At school children were playing with lid of dustbin and this child got hit by it over rt. Side of face. Patient presented to us with swollen right eye, gangrenous upper eyelid. The child was not able to open the eye(right). We immediately sent the lab investigations, took the child to

operation theatre and started debridement. After debridement patient was put on IV antibiotics piperacillin , tazobactum and injection amikacin. Daily dressings were done . Repeated debridements were done as and when required. Wound swab sent for c/s. After ten days swelling subsided 90% ,child was able to open eyes. He was again taken for detailed examination of eye. His visual acuity was normal vision 6/6, ocular movements were normal.

When wound got covered with healthy granulation tissue , and culture was negative for any organism we took the patient for reconstruction. Under GA ,SSG was harvested from his left arm and was used to cover raw area of upper eyelid. Patient was put on IV antibiotics, analgesics. Postoperative period was uneventful. Graft uptake was 100%. Patient was able to open and close the eye and relatives were very satisfied. ***(Figure 7-11)***



Discussion

Necrotising fasciitis also known as flesh eating disease can develop secondary to penetrating trauma or blunt trauma, any skin infection or any ear infection, cervical adenitis or peritonsillary abscess in head and neck region.[9] In 30% of cases generally no precipitating factors are found.[10] Necrosis develops as a result of pathogenic invasion and polymorphonuclear leucocyte infilteration causing vascular thrombosis and ischaemia leading to gangrene of the subcutaneous fat and dermis.

As periorbital skin is very thin and there is paucity of subcutaneous tissue in periorbital area , necrosis occur very early than in other parts of the body.[11] Complications can occur in 66% of cases with periorbital necrotizing fasciitis with mortality of 10%.7 whereas mortality of NF on average ranges between 20% and 35%10,[12] but has been cited as high as 76%.[13]

In 13% of patients visual loss can occur and can be due to orbital spread,corneal perforation or central retinal artery occlusion.[14] If not treated early can lead to death due to septic shock and multiorgan failure.

Management of preorbital necrotizing fasciitis involves aggressive antimicrobial treatment along with early surgical debridement of the necrotic tissue. Fact that thrombosis around the affected site lead to inability of intravenous antimicrobial treatment to reach the tissue reducing its effectiveness. When we surgically debride the wound the number of organisms and toxin load decreases.

In our patients we have done early surgical debridement and given higher IV antibiotics and ended up with positive outcomes.

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Kayser-Fleischer Ring

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Introduction

In the cornea, a golden-brown, ruby-red, or green pigment ring (Kayser-Fleischer ring) consisting of copper deposits appears in peripheral Descemet membrane. Initially thought to be due to the accumulation of silver, they were first demonstrated to contain copper in 1934. Kayser–Fleischer (KF) rings are a common ophthalmologic finding in patients with Wilson disease.

Copper deposition occurs in the posterior Descemet membrane, 1st superiorly, then gradually spreading to meet inferior deposits.

Kayser–Fleischer rings do not cause any impairment of vision but disappear with treatment and reappear with disease progression.

Etiology

Kayser–Fleischer rings are dark rings that appear to encircle the iris of the eye. They are due to copper deposition in part of the Descemet's membrane as a result of liver diseases.

A Kayser-Fleischer ring is found mainly in Wilson disease. It can also be found in primary biliary cirrhosis, chronic active hepatitis, exogenous chalcosis and progressive intrahepatic cholestasis of childhood. These and other non-Wilsonian hepatic disorders can also be associated with Kayser-Fleischer rings, but only Wilson disease is characterized by decreased serum ceruloplasmin levels and neurologic symptoms. It should not be confused with a Fleischer ring, an iron ring seen in keratoconus. Patients with Wilson disease can be differentiated from patients with other diseases that show Kayser-Fleischer rings by their inability to incorporate radioactive copper into ceruloplasmin. K-F-like rings, better termed as bile pigment rings, are seen in majority of patients with high bilirubin and disappear when serum bilirubin levels are below 10 mg/dL.

Epidemiology

Although not all patients with Wilson disease manifest the KF ring but are seen in most of the patients with neurologic involvement from Wilson disease. However, it may not be seen in approximately 5% of these patients. They are present in only 50% of the patients with isolated hepatic involvement and in pre-symptomatic patients. Kayser–Fleischer rings may be absent in patients with fulminant disease and in most children.

Pathophysiology

Kayser–Fleischer rings are caused by deposition of excess copper on the inner surface of the cornea in the Descement membrane extending to the trabecular meshwork. Copper is deposited as a granular complex with sulfur which gives a ring its characteristic color. It is to be noted that copper is present throughout the cornea, however, due to fluid streaming, copper tends to accumulate superiorly and inferiorly, before involving the iris circumferentially. Copper can also be deposited in the lens in the anterior and posterior capsule causing sunflower cataracts which have radiating centrifugal extensions. Both Kayser–Fleischer ring and sunflower cataract do not cause any impairment of vision. Deposition of excess copper in the basal ganglia in the brain leads to the neurologic and psychiatric manifestations of Wilson disease.

Evaluation

Kayser–Fleischer rings do not cause any symptoms. It is usually seen as a golden, brown ring in the peripheral cornea. It starts at Schwalbe's line and extending less than 5 mm onto the cornea. The ring may appear as greenish-yellow, ruby red, bright green, or ultramarine blue. It is almost always bilateral and appears superiorly first, then inferiorly, and then later becomes circumferential. In the initial stages, Kayser–Fleischer rings are usually seen with slit lamp examination; however, as the disease progresses they can be seen with the naked eye, particularly when the iris is lightly pigmented, and there is severe copper overload. Hence, a slit lamp examination is mandatory to make a diagnosis particularly in the early stages unless the rings are visible to the naked eye in conditions of severe copper overload. The slit-lamp assessment commonly used as a standard of care cannot detect them early enough, as the angle view is obscured by the corneal limbus.

Gonioscopy may assist in visualizing the ring even earlier. While the angle can be visualized in gonioscopy, it is not performed routinely, involves applying the three-mirror lens onto the previously anaesthetised cornea and implies full patient co-operation, which makes it difficult for the patients

with neurological symptoms of tremor or dystonic contractures/spasms.

AS-OCT enables imaging of lesions undetectable in a slit lamp for a less experienced diagnostician. It is a noncontact investigative technique, which does not require anaesthetizing the eye, and the scanning duration is below 20 seconds per eye.

Treatment / Management

Once the diagnosis has been established, treatment is given with chelating agents. These agents bind the excess copper in the body and enhance urinary excretion. Two medications used commonly are penicillamine and trientine. Both of these drugs need to be taken orally, and treatment is usually lifelong. Agents like zinc acetate prevent excessive absorption of copper and are also used in conjunction with chelating agents, as maintenance therapy in patients who have been decoppered, and as a temporary measure in pregnancy. The patients should strictly follow a low copper diet. Patients who present with acute liver failure will need emergent liver transplant evaluation as medical treatment alone is ineffective in such patients.

Kayser–Fleischer rings typically disappear with treatment and reappear with disease progression. When present, it a useful clinical sign to monitor treatment compliance. The rings disappear with chelation therapy over three to five years in up to 80% of patients

WILSON DISEASE

PATHOGENESIS Wilson disease (hepatolenticular degeneration) is an autosomal recessive disorder caused by multiple allelic substitutions or deletions in a Cu2+-ATPase-transporting β-polypeptide, linked to mutations in the ATP7B gene on chromosome 13 (13q14.3–q21). Copper is deposited 1st in the liver, then in the kidneys, and eventually in the brain and the cornea at the Descemet membrane.

CLINICAL PRESENTATION Muscular rigidity increases, and tremor and involuntary movement gradually occur in a fluctuating course resembling parkinsonism. Unintelligible speech and mild dementia usually occur concomitantly. Equal numbers of patients (40%) present with hepatic or nervous system symptoms. A "sunower" cataract may be present.

History and Physical Examination

Wilson Disease should be suspected in all patients with the following:

- Kayser–Fleischer rings detected on routine eye exam
- A sibling or parent with a diagnosis of Wilson disease
- Unexplained and acquired Coombs-negative hemolytic anemia
- Neurological symptoms of unexplained origin
- Psychiatric disease with signs of hepatic or neurologic disease
- In persons between ages three and 55 years with unexplained serum aminotransferase elevation, chronic hepatitis with steatosis, poorly responsive autoimmune hepatitis, cirrhosis, or acute liver failure.

Age alone should not be the used as a criterion to disregard a diagnosis of Wilson disease. A few patients older than 55 years present with neurologic symptoms of Wilson disease, particularly when such symptoms were overlooked.

LABORATORY EVALUATION Low serum ceruloplasmin, high non-ceruloplasmin-bound serum copper, and high urinary copper suggest the diagnosis, which can be confirmed with liver biopsy. Nonspecfic findings of proteinuria, aminoaciduria, glycosuria, uricaciduria, hyperphosphaturia, and hypercalciuria are seen. The cranial magnetic resonance shows hyperintense, bilateral and symmetric images in the basal ganglia and brainstem.

MANAGEMENT

Wilson disease is usually managed by a multidisciplinary team that includes an ophthalmologist, neurologist, gastroenterologist, and a hepatologist. The outpatient management is by the nurse practitioner and primary care provider Wilson disease can be treated with penicillamine. Liver transplantation is reserved for patients with fulminant liver failure. The Kayser-Fleischer ring disappears gradually with therapy, including liver transplant, and the disappearance of the rings can be used to help monitor therapy. Electrophysiologic abnormalities from retinal dysfunction have been shown to reverse after treatment of the disease.

Pearls and Other Issues

Wilson disease is an autosomal recessive disease where the affected organs exhibit elevated copper levels due to copper toxicity. Intestinal copper absorption is not increased in patients with Wilson disease, but the biliary excretion of copper is reduced. The reduced biliary excretion of copper is possibly due to a defect in the entry of copper into lysosomes, but the delivery of the lysosomal copper to bile is intact. Although serum ceruloplasmin is low in patients with Wilson disease, this finding is unlikely to be responsible for the copper toxicity seen in Wilson disease. The low ceruloplasmin is due to the lack of incorporation of copper into apoceruloplasmin, which has a shorter half-life than copper-bound ceruloplasmin (holo-ceruloplasmin).

Clinical symptoms of Wilson disease are rarely present before five years of age, and most untreated patients manifest with symptoms by the age of 40. Low concentration of ceruloplasmin in serum can be due to many causes, such as severe liver disease causing diminished synthesis, nephrotic syndrome, protein-losing enteropathy, and intestinal

malabsorption. Normal levels of serum ceruloplasmin do not rule out Wilson disease. This is seen in at least 15% of patients with Wilson disease and is usually seen as an acute phase reactant to severe liver injury due to any cause, and in patients with elevated serum estrogen levels.

In summary, the presence of Kayser–Fleischer ring or significantly elevated hepatic copper concentration obtained via a liver biopsy and a low serum ceruloplasmin level establishes the diagnosis of Wilson disease in all cases where the diagnosis is suspected.

Kayser–Fleischer rings are a sign of Wilson disease. They are not specific to Wilson disease alone. Kayser–Fleischer rings rarely are seen in other chronic cholestatic disorders such as primary biliary cholangitis and children with neonatal cholestasis.[4]

As per the American Association for Study of Liver Disease guidelines, the presence of Kayser–Fleischer rings, low serum ceruloplasmin concentration (less than 20 mg/dL), and elevated basal 24-hour urinary excretion of copper are sufficient to establish a diagnosis of Wilson disease. If Kayser–Fleischer rings are present but the serum ceruloplasmin is above 20 mg/dL, a liver biopsy is needed to make a diagnosis of Wilson disease. In the absence of Kayser–Fleischer rings, a liver biopsy with hepatic copper quantification is mandatory to confirm the diagnosis of Wilson disease. Normal urinary excretion of copper is less than 40 micrograms in 24 hours. Most patients with Wilson disease have urinary copper excretion higher than 100 micrograms in 24 hours, and the copper excretion often exceeds 1000 micrograms per 24 hours in those with fulminant hepatic failure. A hepatic copper concentration of more than 250 micrograms per gram of dry weight, accompanied by low serum ceruloplasmin and elevated basal 24-hour urinary excretion of copper establishes the diagnosis of Wilson disease. In cases where the hepatic copper content is between 50 and 250 micrograms per gram of dry weight, molecular testing (confirming homozygosity for one mutation or two mutations constituting compound heterozygosity) is required to make a diagnosis of Wilson disease. If the hepatic copper content is less than 50 micrograms per gram of dry weight, a diagnosis of Wilson disease is excluded. An elevated copper concentration alone on liver biopsy does not make a diagnosis of Wilson disease. Hepatic copper concentration may be elevated in other chronic cholestatic disorders and non-Wilsonian hepatic copper overload conditions such as Indian childhood cirrhosis, Tyrolean infantile cirrhosis, and idiopathic copper toxicosis.

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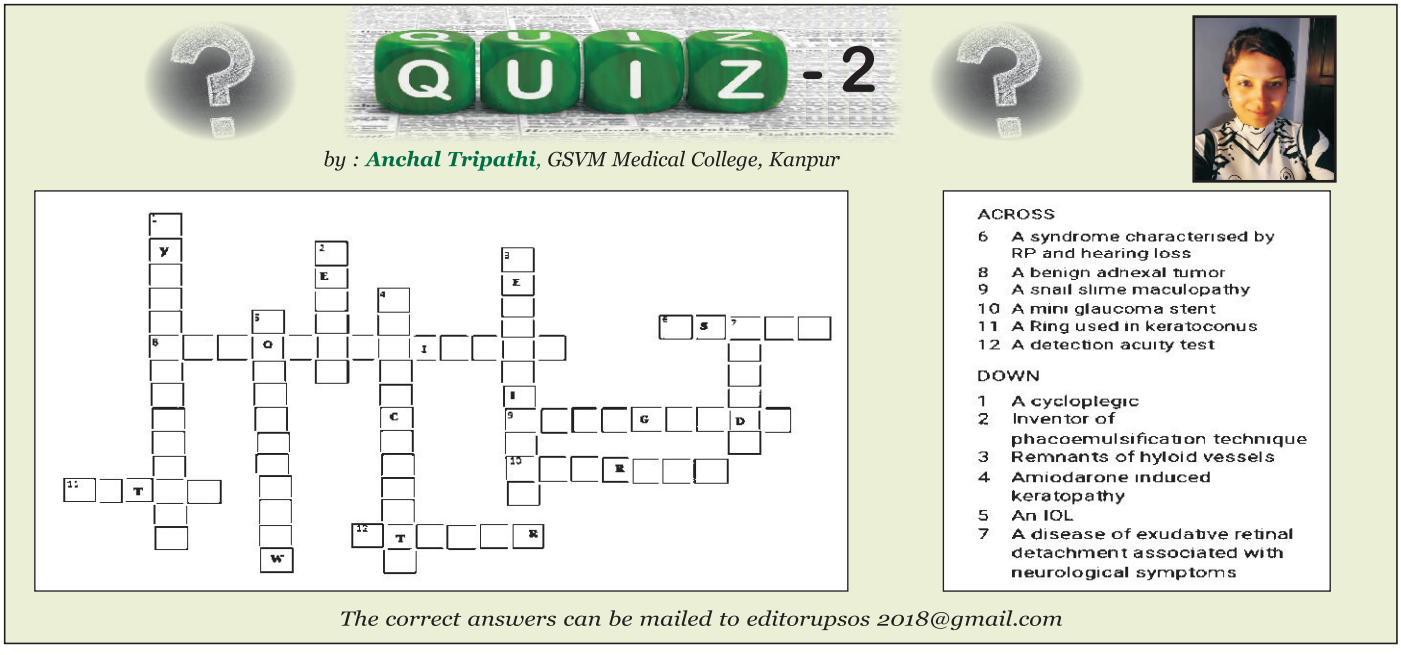

Digital Visual Fatigue – Are We Prepared?

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ABSTRACT:

Digital visual fatigue or computer vision syndrome is becoming one of the common issues to be dealt by an eye specialist. The symptoms, identification of signs, reasons and methods to prevent ocular damage are the need of the hour. Today millions of children also use computers /smart phones on a daily basis. Very few studies are available to investigate the impact of digital devices on children eyes. This article aims to discuss the symptoms, reasons and methods to prevent computer vision syndrome and a tailored approach to attend the children getting affected on a regular basis.

KEY WORDS: Computer vision syndrome, e –readers, management

Symptom Category	Symptoms	Possible Cause
Asthenopic	Eye strain, tired eyes, sore eyes	Binocular vision, accommodation
Ocular surface related	Dry eyes, watery eyes, contact lens problems	Decreased blink rate (from normal 22/min to 7-10/min)
Visual	Blurred vision , slowness in focus change, double vision, presbyopia	Refractive error, accommodation, binocular vision ,presbyopia correction
Extra ocular	Neck pain, back pain, shoulder pain	Computer screen location

According to International Classification of Diseases (ICDS 10) of WHO Visual fatigue or strain is defined as-A degree of visual discomfort typically occurring after some kind of prolonged visual activity and characterised by fatigue, pain around eyes, blurred vision and headache.

SYMPTOMS:

In case of digital visual fatigue, the symptoms can be categorized into four major groups

Main Reasons –

1) Due to Reflection and Glare from screen- When the screen is positioned in front of a window or a light source, it reflects of screen. As we move around (in case of mobile) glare changes and eyes again have to adjust for different lightings.

2) Due to screen flicker while refreshing screen- Every digital screen has to be refreshed because they do not have the capacity to hold a stable image on the screen. They just give illusion of a constant image. Usually the refresh rates of LCD and LED monitors are 125 to 250 Hz. In case of CRT monitors it was 50 to 60 Hz. Increase in refresh rates leads to decrease in image blur. Refresh rates of most of the mobile screens is around 60 Hz.

3) Due to PWM Brightness control- Brightness of the digital display can be altered to match both dim and bright environments. Traditional method to decrease brightness of display is Pulse Width Modulation (PWM) which works by rapidly cycling on and off of backlights. (Usually at frequency of 180 to 240 Hz). This may cause screen flicker and visual fatigue.

4) Due to HEV Blue light - Digital displays emit High Energy Visible (HEV) Blue light that can lead to macular degeneration. These lights are also emitted by sun and LED bulbs but we don't stare at them!

5) Due to reduced working distance in monitors and smart phone as compared to paper print- Typical near working distance for paper print is 35 to 40 cm while for internet viewing it is average 32cm (19 to 60 cm) and for text message on smart phone is 36 cm (17.5 to 58 cm). This decreased working distance increases demand of accommodation and vergence (By 0.5 to 0.75 D).

Eye Strain Reduction Techniques-

1) *Anti-reflective coating and Glass colour tinting*- Present day monitors have anti-reflective coating to decrease glare while facing a light source. Glass colour tinting decides how much of the visible light reaches our eyes and how well we see other colours and contrast. Rose/Red colour tinting is said to increase contrast and block blue light. Many people feel that rose tinted lenses are more comfortable for long period of time

2) *Fuzzy Logic based Brightness control*- Automatic control of brightness is preferred where fuzzy controller takes intensity of atmospheric light (measured by a sensor) and

current screen brightness as input and modifies the screen brightness accordingly.

3) *Optimizing monitor's colour temperature-* Colour temperature affects picture quality and colour reproduction. If colour temperature is low, white objects appear redder. If colour temperature is higher, white objects appear blue. Open a blank white screen and adjust colour temperature of display to match colour temperature of environment. This will help reduce eye strain

Sunlight	*Colour Temperature*	*Artificial Light*	*Colour Temperature*
Clear sky	12000K	Day light Flourescent	6500K
Cloudy sky	6500K	White Flourescent	4200K
Average noon sunlight	5300K	Soft white Flourescent	3000K

4) *Good practices to reduce eye strain-*

i) Maintain a comfortable working distance (ONE , TWO, TEN) One foot for mobile phones, Two feet for desktop and Laptop devices and Ten feet for TV screens.

ii) 20-20-20 rule- after every 20 minutes of work, look 20 feet away for 20 seconds

iii) Lighting levels should be 200 to 700 lux

iv) All screens and monitors should be dust free as dust causes glare

v) Refocusing from screen to print and vice versa should be avoided by using document holders attached to screen

vi) Artificial tear substitutes tend to relieve the symptoms of digital visual fatigue to some extent.

vii) Frequent voluntary blinking should be practised while working long hours on computer.

E-readers and Visual Fatigue –

E- Readers (KINDLE etc.) don't use light to light up pixels but rather E- Ink. They have electronic ink laminated to a plastic film substrate. E-Ink is simply arranged on the screen where pixels are and this creates image. E –ink readers display is "Reflective" i.e.no back light is used and ambient light from the environment is reflected from the surface (LCD display is "Emissive" i.e. light from a backlight is projected through a display towards eye).

So E –Readers don't need any light to lit up; no changes in contrast that eyes have to adjust. Also no glass to cause reflection or glare. Thus reading on an E reader is actually same as reading a paper.

Then why are we not using E – ink screens for computer and other devices?

i) They take long time to change screen. So they cannot support video or computer games.

ii) They are currently available only in white and black colour although research is still on.

How are our children getting affected??

Today millions of children use computers /smart phones on a daily basis. Very few studies are available to investigate the impact of digital devices on children eyes. Children can experience many of the same symptoms as an adult. Some unique aspects of child using a digital device are-

1) Limited degree of self awareness- Most of the kids keep continuing their task, (eg. Playing games on mobile) till they get exhausted with very few breaks. So accommodation is "Locked in" to a particular target distance that can sometimes lead to accommodative spasm. They can also suffer from eye irritation (poor blinking causing poor tear film distribution).

2) Adaptability of children- A child using a computer or mobile with large amount of glare often will not think of changing the settings to make viewing more comfortable. Kids often accept blurred vision caused by refractive error because they think everyone sees the way they do.

3) Using an adult computer- A child sitting on father's computer has to look up much further than his father. Kids also face issues while reaching for keyboard causing neck , arm or back pain.

Mobile phone dependency is leading to negatively predicted attention, positively predicted depression, affect on social relations and academicachievements.

The child does not get enough sleep and remains irritable affecting his social development.

What should we do?

1) Regular eye examinations- at least annual including refraction and orthoptic exercises

2) Decrease the time in which child uses computer- ask for 10 minutes break after every one hour

3) Check position of computer- Screen should not be too high. Opt for adjustable chair, foot stool etc

4) Check lighting for glare on screen- windows and other light sources can cause glare; computer screen should be turned in other direction

5) No need to introduce technology too early , at least not before 3 years(Avoid giving VIRTUAL PACIFIERS OR ELECTRONIC BABY SITTERS)

6) Avoid exposure to devices at least 1 hour before sleep

7) Inculcate good habits like keeping meal time or parent –kid play time free and unplugged

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Minimally Invasive Approach for Malignant Glaucoma in Pseudophakic Eyes : A Simple Technique for A Sight Threatening Situation

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Abstract:

Malignant glaucoma due to aqueous misdirection is one of the most challenging diagnostic and therapeutic situations. Timely intervention can save the eye from blindness.

This case series illustrates some of the important features and problems that may be encountered during the management of malignant glaucoma. Although eyes with malignant glaucoma may initially respond to medical management, long term cycloplegia is usually necessary to maintain resolution.

Sensitisation to atropine drops is not uncommon and may occur at any time necessitating alternative treatment. And surgical treatment, either a needle aspiration of vitreous through the pars plana or pars plana vitrectomy involves cost, discomfort and risk of complications.

Traditionally, management of malignant glaucoma involves pars plana vitrectomy to rupture the anterior hyaloid face and relieve the aqueous misdirection. Peripheral laser Nd YAG Capsulotomy outside the optic edge is a cost effective and a definitive treatment for capsular malignant glaucoma , It creates a communication between the vitreous and anterior chamber thus vitrectomy which is a more hazardous approach specially in eyes with advanced glaucomatous optic nerve head damage can be avoided. We present a series of three cases of malignant glaucoma that occurred post trabeculectomy in pseudophakic eyes and were conservatively managed using Nd:YAG laser anterior hyaloidotomy with immediate deepening of the anterior chamber and long term control of intraocular pressure.Till recent follow up there was no recurrence of aqueous misdirection.

Introduction

Malignant glaucoma continues to present a difficult diagnostic and therapeutic challenge to the ophthalmologist. The treatment of malignant glaucoma involves rupturing the anterior hyaloid face to relieve the aqueous misdirection.[1] Surgical intervention in the form of pars plana vitrectomy is the most frequently described technique to achieve this.[2] We describe a series of cases where a more conservative, previously defined technique was used to relieve the aqueous misdirection using the Nd:YAG laser.

Case 1 :

A 48year old lady pre¬sented with complaints of sudden pain and blurring of vision in the left eye. She gave history of undergoing Trabeculectomy for angle closure glaucoma in the left eye2 years back and Phacoemulsification with IOL implantation 6 months back. On examination, left eye showed very uniformly shallow anterior chamber involving both peripheral and central anterior chamber (figure 1A).Intraocular pressures (IOP) were 20 mm Hg in right eye and 44 mm Hg in left eye.

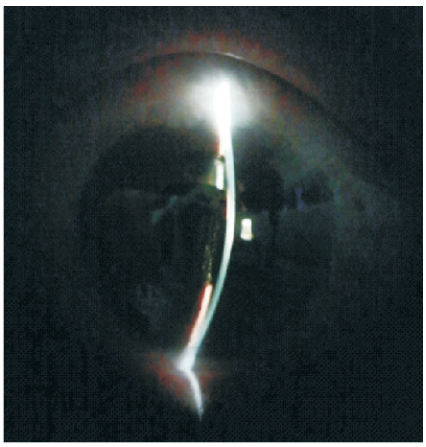

Figure 1A:

Uniformly shallow anterior chamber both centrally and peripherally

In view of a patent peripheral iridotomy and above findings, a diagnosis of malignant glaucoma was made. The patient was treated with topical and oral glaucoma medications and atropine eye drops following which the IOP dropped to 18mmHg.

During the following 3 months the IOP was maintained below 20 mm Hg on atropine once daily. However, once atropine was withdrawn due to allergic reactions, the left eye soon developed recurrence of pain and blurred vision, an IOP of 38mmHg and the same clinical picture of malignant glaucoma.

We performed Nd Yag capsulotomy and anterior hyaloidotomy through the previous patent peripheral iridotomy (Figure 1B) and through the capsule outside the IOL optic edge(Figure 1C) following which the chamber deepened immediately, both centrally and peripherally (Figure 1D) and IOP dropped to 12 mHg.

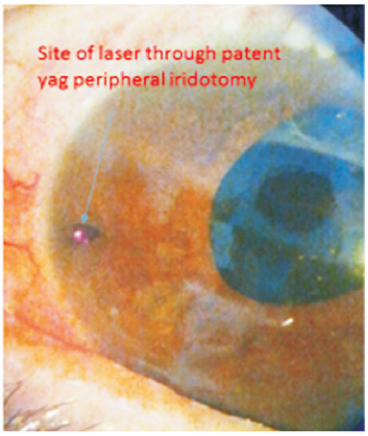

Figure 1B : Site of laser anterior hyaloidotomy

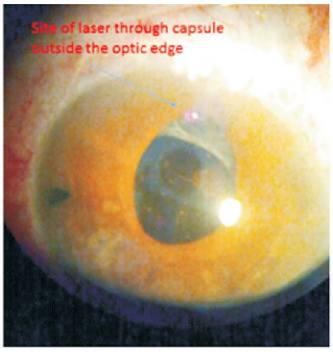

Figure 1C: Site of laser anterior hyaloidotomy

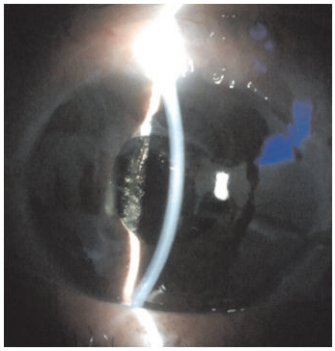

Figure 1D: Deepening of anterior chamber after laser anterior hyaloidotomy

Patient was symptomatically relived,the deep anterior chamber was maintained and IOP was controlled up to two years follow up.

Case 2:

A 20 year old girl presented to us with loss of vision in both eyes. Her vision was 6/36 in Right Eye and 1/60 in Left Eye. She gave history of trabeculectomy in both eyes two years back. Her IOP was 42mmhg in right eye and 14 mmhg in left Eye with maximal medical therapy. Slit lamp evaluation showed anterior chamber of variable depth in the right eye due to posterior synaechiae and complicated cataract. The Cup Disc Ratio was 0.85 in right eye and 0.9 in the left eye and gonioscopy showed closed angles. She underwent repeat trabeculectomy combined with phacoemulsification and IOL implantationin the right eye (Figure 2A). Three weeks after surgery she developed very shallow anterior chamber (Figure 2B), very high IOP (38 mmHg) and myopic shift in refraction caused by forward movement of lens iris diaphragm. Malignant glaucoma was diagnosed and YAG capsulotomy and anterior hyaloidotomy was performed outside the optic edge (Figure 2C) following which the anterior chamber formed immediately (Figure 2D) and IOP was contolled (14 mmHg).

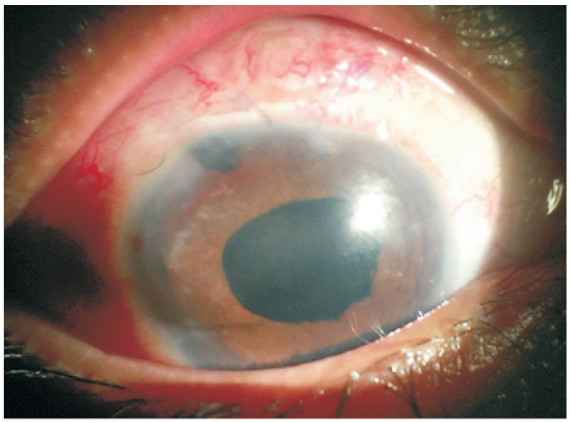

Figure 2A: Post Trabeculectomy with IOL implantation

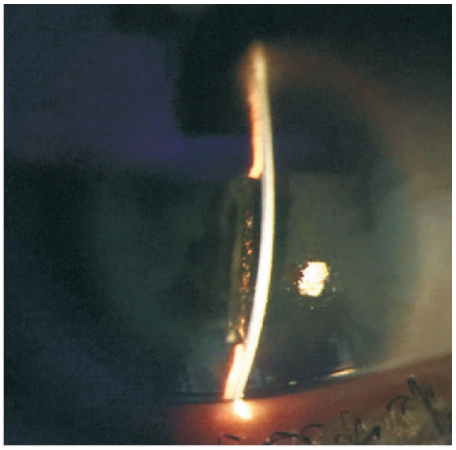

Figure 2B: Uniformly very shallow AC

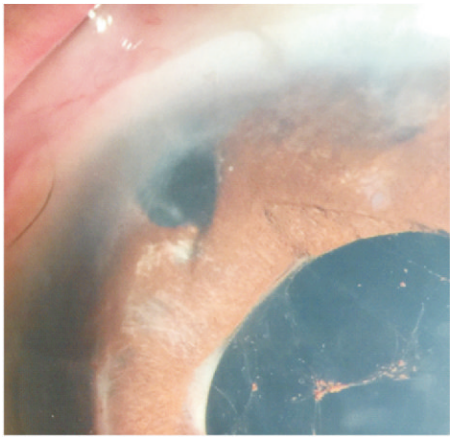

Figure 2C: Capsulotomy with anterior hyaloidotomy done via iriditomy peripheral to the IOL optic edge

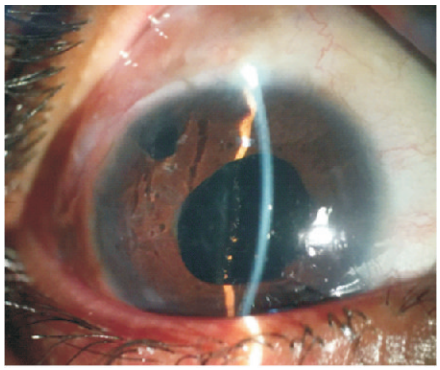

Figure 2D: Immediate deepening of the anterior chamber

Case 3:

A 56 year old lady presented to us with BCVA of 6/18 in Both Eyes and IOP of 24mmHg in right eye and 40 mmHg in the left Eye with maximal medical therapy. Slit lamp evaluation showed shallow anterior chamber with patent YAG PI and posterior subcapsular cataract in Both Eyes. Both eyes had advanced cupping. The left eye underwent combined phacoemulsification surgery with trabeculectomy. One month after surgery, the left eye developed malignant glaucoma with uniform shallow AC (Figure 3A) and very high IOP (42 mmHg). We performed YAGcapsulotomy and anterior hyaloidotomy outside the optic edge (Figure 3B,C) following which the anterior chamber formed immediately (Figure 3D) and IOP returned to 12 mmHg.

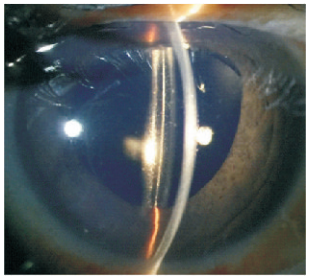

Figure 3A:

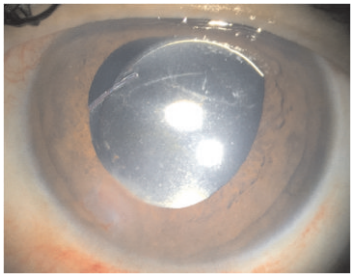

Figure 3B:

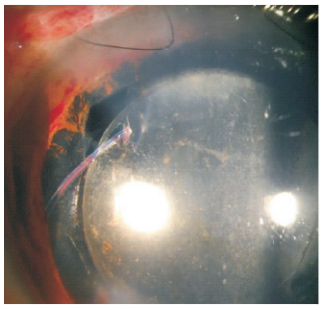

Figure 3C:

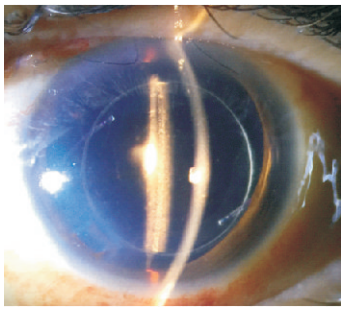

Figure 3D:

Discussion

We present a case series of malignant glaucoma that developed in eyes that had pre-existent angle closure glaucoma and had all undergone trabeculectomy. The diagnosis was based on the findings of very high IOP and uniform shallowing of both the central and peripheral anterior chamber in an eye with a patent iridectomy as opposed to pupillary block where the peripheral chamber is much shallower than the central anterior chamber.

Management of malignant glaucoma requires reversal of the aqueous misdirection from the anterior vitreous back into the anterior chamber by creating a conduit. This can be done by rupturing the anterior hyaloid face peripheral to the IOL optic circumference in pseudophakic eyes using a Nd:YAG laser to rupture the posterior capsule as well as the anterior hyaloids.3 However the channel created must be peripheral to the IOL optic to be effective . One way of improving the likely outcome of Nd:YAG laser therapy is to make the capsular opening through a dialing hole in the IOL or through a pre-existent iridotomy where present, as we have shown, thus allowing a direct passage of aqueous between vitreous cavity and anterior chamber. Making an opening in the anterior hyaloids immediately posterior to the IOL optic is futile as effective passage of aqueous into the anterior chamber is impeded by the IOL itself.

There have been several reports in the past showing successful laser treatment to resolve malignant glaucoma, similar to what we describe.[4,5,6]

In a series of three cases, Melamed et al reported successful reversal of malignant glaucoma and instant deepening of the anterior chamber with marked drop in IOP following Nd:YAG hyaloidotomy.[7] The authors also suspect that use of large optic (7 mm) posterior chamber lens implants may increase the risk of developing malignant glaucoma postoperatively and may also present an obstacle to successful hyaloidotomy as they may prevent adequate flow of aqueous from the vitreous into the anterior chamber.

Our experience reiterates the fact that such non invasive therapy should be attempted in all cases prior to invasive treatment such as needle aspiration of vitreous or surgical pars plana vitrectomy, keeping in mind that the hyaloidotomy should be peripheral to the IOL optic. Additionally, the IOP reduction following the procedure is sustained over prolonged periods of time as seen from our experience.

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7. Melamed S, Ashkenazi I, Blumenthal M. Nd-YAG laser hyaloidotomy for malignant glaucoma following one-piece 7 mm intraocular lens implantation. Br J Ophthalmol. 1991;75:501–503.

Scientific Programme of Mid-Term UPSOS Conference

25-26th May 2019
MRA Medical College,Ambedkarnagar,UP

DAY -1
25/05/2019 (SATURDAY)

DELEGATE REGISTRATION(ON SPOT)
Time : 11.00 AM - 12.00 PM

SURGICAL SKILLS TRANSFERCOURSES
Time : 12.00 - 2.00 PM

LUNCH SESSION
Time : 02.00 PM - 03.00 PM

SESSION 1
Convener-Dr Durgesh & Dr Nevendu Rai
Courses: Phaco and SICS
Time: 3.00 PM - 3.30 PM

LESSONS FROM THE MASTER

Chairman : Prof. S P Singh
Co- Chairman : Dr. Dharmendra Nath
Convenor : Dr. Anil Kumar Srivastava
Moderator : Dr. Amit Kumar Patel

Dr Partha Biswas

1. Phaco in Small Pupile
2. IOL Placement in PCR

PG PAPER SESSION
Time: 3.30 PM - 6.20 PM

Judges: Prof. K J Singh, Prof. Madhu Bhadauria, Prof. R C Gupta and Dr. Deepak Mishra

Topics **Talk duration: 08 Minutes**

S. No.	Title	Speaker
1	Prevalence of Dry Eye Disease in Post Menopausal Women:A teaching hospital survey. **(3.30 PM - 3.38 PM)**	**Dr. Anurag Kumar Kashyap** Dr. Rajendra P Maurya Dr. Virendra Pratap Singh Dr. Tanmay Shrivastava
2	Evaluate foldable hydrophilic and hydrophobic acrylic IOLs implantation in pediatric cataract surgery **(3.38 PM - 3.46 PM)**	**Dr. Sarswati** Dr R Y S yadav
3	Corneal Endothelium Changes After Small-Incision Cataract Surgery in Patients With Diabetes Mellitus **(3.46 PM - 3.54 PM)**	Dr. Rishi Tripathi
4	Approach to diagnosis and management of diabetic retinopathy **(3.54 PM – 4.02 PM)**	**Dr. Rajesh kumar**
5	Comparison of change corneal astigmatism in pre and post operated pterygium excision **(4.02 PM – 4.10 PM)**	**Dr. Harish Kumar**
6	Glaucoma in Females- Anaemia a risk factor **(4.10 PM – 4.18 PM)**	**Dr. Anupriya** Dr. Sunil Kumar Prof S K Bhasker
7	Blueberry eye : after fungal corneal ulcer a case report of acquired total anterior staphyloma, a rare anterior segment pathology **(4.18 PM – 4.26 PM)**	**Dr. Sameeksha Agrawal** Dr. Ankit Agrawal Dr. K K Agrawal Dr. V.K Agrawal
8	Spontaneous bilateral subluxation of PCIOL after 10 years due to pseudoexfoliation syndrome **(4.26 PM – 4.34 PM)**	**Dr. Ankit Agrawal** Dr. Sameeksha Agrawal Dr. K K Agrawal Dr. V K Agrawal
9	Macular thickness Changes assessment by Spectral Domain OCT(SD-OCT) following extracapsular cataract extraction(ECCE) **(4.34 PM – 4.42 PM)**	**Dr. Samreen Mehfooz**
10	Clinical study of ACIOL and sclerafixated PCIOL **(4.42 PM – 4.50 PM)**	**Dr. Aishwarya Madharia**
11	To evaluate biomechanical properties of cornea in thyroid ophthalmopathy **(4.50 PM – 4.58 PM)**	**Dr. Shailja mishra**
12	To analyse and study the outcome of benign cyctic orbital lesions treated with foam Sclerotherapy **(4.58 PM – 5.06 PM)**	**Dr. Stuti Tiwari**
13	Trojan horse anaesthesia: A novel method of anaesthesia for pars plans vitrectomy **(5.06 PM – 5.14 PM)**	**Dr. Ritu Singh** Prof. Sanjiv Kumar Gupta Dr. Ajai Kumar Dr. Arun Sharma
14	To study the role of medical management and their outcome in extra-ocular cysticercosis with the role of additional use topical cyclosporine. **(5.14 PM – 5.22 PM)**	**Dr. Manmeet Singh**
15	Trans scleral fixation of closed loop haptic acrylic posterior chamber intraocular lens in aphakic non vitrectomized eyes **(5.22 PM – 5.30 PM)**	**Dr. Divya Gupta** Dr. Sanjiv Kumar Gupta Dr. Siddharth Agrawal
16	To evaluate effect of intravi treal injection Ranibuzumab on CME due to Retinal vein occlusion **(5.30 PM – 5.38 PM)**	**Dr. Anil**
17	Prospective study of effectiveness of intrastromal Voriconazole injection in MGT of deep non healing fungal corneal ulcer **(5.38 PM – 5.46 PM)**	**Dr. Ashutosh**
18	Prospective outcome of Single suture on SIA in SICS **(5.46 PM – 5.54 PM)**	**Dr. Praveen Chaturvedi** Dr. Diksha Prakash Prof. OPS Maurya
19	Epidemiology of Corneal Ulcer in North India **(5.54 PM – 6.02 PM)**	**Dr. Hemendra Singh** Dr. Prashant Bhushan
22	How to write paper-Tips for PGs **(6.02 PM – 6.20 PM)**	**Dr.Deepak Mishra** *(Key Note Speaker)*

RETINA SESSION

Time: 6.20 PM - 7.30 PM

Chairman : **Dr. Prashant Bawankule**
Co- Chairman : **Prof. Neelima Mahlotra**
Convenor : **Dr. Mohita Sharma**
Moderator : **Dr. Satya Prakash Tiwari**

Talk duration : 10 Minutes
Key Note speaker : 15 Minutes

Topics

S.No.	Title	Speaker
1	3D vitrectomy for PDR **(6.20 PM - 6.35 PM)**	**Dr. Prashant Bawankule** *(Key Note Speaker)*
2	Managing retinal disorder with coexisting corneal pathology **(6.35 PM - 6.50 PM)**	**Dr. Satya Prakash Tiwari** *(Key Note Speaker)*
3	Retinal evaluation for general ophthalmologist **(6.50 PM - 7.00 PM)**	**Dr. Navendu Rai**
4	Diabetic macular odema,stepwise approach to treat **(7.00 PM - 7.10 PM)**	**Dr. Abhishek Dixit**
5	Diabetic Maculopathy **(7.10 PM - 7.20 PM)**	**Dr. Durgesh Srivastava**
6	Phaco in vitrectomised eyes **(7.20 PM - 7.30 PM)**	**Dr. Prashant Bawankule**

GBM/EXECUTIVE & SCIENTIFIC COMMITTEE MEET

Time: 7.30 PM -8.15 PM

GALA DINNER

Time: 08.15 PM Onwards

DAY -2

26/05/2019 (SUNDAY)

CATARACT SESSION

Time: 8.00 AM - 9.10 AM

Chairman : **Prof. D J Pandey**
Co- Chairman : **Dr. Dharmendra Nath**
Convenor : **Dr. Shashi Bhasker**
Moderator :**Dr. Shalini Mohan**

Talk duration: 10 Minutes

Topics

S.No.	Title	Speaker
1	Role of tear film in cataract and Lasik surgery **8.00 AM - 8.08 AM**	Dr.Anil kumar Srivastava
2	Cataract surgery in post PK patients **8.10 AM - 8.20 AM**	Dr. Shalini Mohan
3	Toric IOLs **8.20 AM - 8.30 AM**	Dr. Dharmendra Singh
4	Zero volume Phaco **8.30 AM - 8.40 AM**	Dr. Dharmendra Nath
5	Optimizing results with multifocal/trifocal IOLs **8.40 AM - 8.50 AM**	Dr. Mohita Sharma
6	Managing corneal odema after cataract Surgery **8.50 AM - 9.00 AM**	Dr. Namrata Sharma
7	IOL Power calculation in different situations **9.00 AM - 9.10 AM**	Dr. Eram Praveen

Short Break

INAUGURAL FUNCTION

Time: 9.20 AM - 10.30 AM

CORNEA & REFRACTIVE SESSION

Time: 10.30 AM - 1.20 AM

Chairman : **Prof. Namrata Sharma**
Co- Chairman : **Prof. S.P.Singh**
Convenor : **Prof. OPS Maurya**
Moderator : **Prof. A K Jaiswal**

Talk duration: 10 Minutes
Key Note speaker: 20 Minutes

Topics

S. No.	Title	Speaker
1	Complications of LASIK Surgery **(10.30 AM - 10.50 AM)**	**Dr. Namrata Sharma** (Key Note Speaker)
2	Administrative, Social and legal issues related to Eye Donations and Corneal transplantation **(10.50 AM - 11.00 AM)**	**Dr. R C Gupta**
3	ARC for You **(11.00 AM - 11.20 AM)**	**Dr. Partha Biswas** (Key Note Speaker)
4	Advances in Keratoconus **(11.20 AM - 11.30 AM)**	**Dr. D J Pandey**
5	Refractive lens exchange **(11.30 AM - 11.40 AM)**	**Dr. K J Singh**
6	Corneal aberrations-basic **(11.40 AM - 11.50 AM)**	**Dr. Shashi Bhashkar**
7	Relevance of human analogue to Artificial Inteligence **(11.50 AM - 12.00 PM)**	**Dr. Malay Chaturvedi**
8	Dry eye **(12.00 PM - 12.10 PM)**	**Dr. Jimmy Mittal**
9	Loop Myopaxy For Myopic Strabismus Fixus **(12.10 PM - 12.20 PM)**	**Dr. S.K.Sharma**
10	Management of Keratoconous **(12.20 PM - 12.30 PM)**	**Dr. Bhavesh Makkar**
11	Workup of Fungal Ulcer **(12.30 PM - 12.40 PM)**	**Dr. O P S Maurya**
12	Graft Rejection **(12.40 PM - 12.50 PM)**	**Dr. Azad Gaurav Bansal**
13	Corneal Dystrophy **(12.50 PM - 1.00 PM)**	**Dr. Pankaj Baranwal**
14	Keratoprosthesis **(1.00 PM - 1.10 PM)**	**Dr. Srikant**
15	Approach to tackle uncommon corneal cases **(1.10 PM - 1.20 PM)**	**Dr. Arushi Goyal**
16	How to manage yourself and practice **(1.20 PM - 1.30 PM)**	**Dr. Khurseed Khan**

SURGICAL SKILLS TRANSFER COURSES

Time: 1.30 PM - 2.45 PM

SESSION 2

Convener-Dr Eram Praveen & Dr Shashi Bhaskar
Courses: Phaco and SICS

LUNCH SESSION

Time: 02.45 PM - 03.30 PM

VALEDICTORY

Time: 03.30 PM - 04.00 PM

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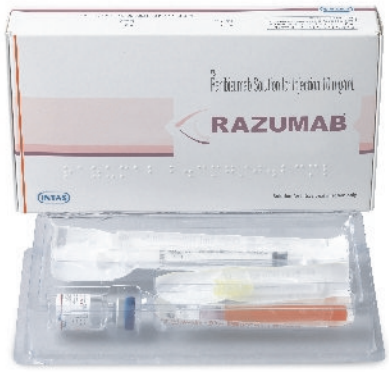

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1. Myopic Choroidal Neovascularization

*Internal Data

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